ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY

FOUNDED BY JAMES PLEASANT PARKER

VOLUME 51

Editors

L. W. DEAN, M.D.,
1125 N. Berry Road, Kirkwood, Mo.

ARTHUR W. PROETZ, M.D.,
Beaumont Building, St. Louis

Editorial Board

T. E. CARMODY, M.D. . . . . *Denver*
IRA FRANK, M.D. . . . . *Chicago*
W. E. GROVE, M.D. . . . . *Milwaukee*
DAVID HIGBEE, M.D. . . . . *San Diego*
ANDERSON C. HILDING, M.D. . . *Duluth*
FREDERICK T. HILL, M.D. . *Waterville, Me.*
MARVIN F. JONES, M.D. . . . . *New York*
HAROLD I. LILLIE, M.D. . *Rochester, Minn.*
JOHN G. MCLAURIN, M.D. . . . . *Dallas*
LEROY A. SCHALL, M.D. . . . . *Boston*
H. MARSHALL TAYLOR, M.D., *Jacksonville, Fla.*
O. E. VAN ALYEA, M.D. . . . . *Chicago*

Published Quarterly

BY THE

ANNALS PUBLISHING COMPANY

EDITORIAL OFFICE — 1010 BEAUMONT BLDG.
BUSINESS OFFICE — 7200 WYDOWN BL.

ST. LOUIS, MO., U.S.A.

Annual Subscription in United States, Spain, Central and South America, $6.00 in Advance. Canada, $6.40. Other Countries, $6.80.

Contents.

PAGE

Harris Peyton Mosher 583

LII.—Laryngocele. Charles H. Allman, M.D., and David P. Cordray, M.D., Boston 585

LIII.—Deep Neck Infection. August L. Beck, M.D., New Rochelle, N. Y. 592

LIV.—Acute Mastoiditis: 1931-1941. Lawrence R. Boies, M.D., Minneapolis 601

LV.—Intranasal Application of Sulfonamide Powder. Lester A. Brown, M.D., Atlanta, Ga. 611

LVI.—Bilateral Jugular Ligation Following Bilateral Suppurative Mastoiditis. Maurice G. Evans, M.D., Boston 615

LVII.—The Significance of the Nose in Allergic States. Ralph J. Frackelton, M.D., Lakewood, Ohio 626

LVIII.—Acute Fulminating Osteomyelitis of the Frontal Bone: A Review and a Prediction. Gustave B. Fred, M.D., Boston 638

LIX.—Some Causes for Failure in Frontal Sinus Surgery. Robert Lincoln Goodale, M.D., Boston 648

LX.—The Changing Conception of the Management of Chronic Progressive Deafness. Frederick T. Hill, M.D., Waterville, Me. 653

LXI.—Dermoid Cysts of the Nose. Edgar M. Holmes, M.D., Boston 662

LXII.—A Cooperative Bronchoscopic Clinic. Gordon D. Hoople, M.D., Syracuse, N. Y. 671

LXIII.—Ménière's Disease. Charles I. Johnson, M.D., Boston 676

LXIV.—Air Pressures in the Nose and Maxillary Sinus Under Normal Conditions in Disease. Delbert K. Judd, M.D., Chicago 689

LXV.—The Treatment of Congenital Atresia of the Choanae. V. H. Kazanjian, D.M.D., M.D. 704

LXVI.—The Degeneration and Absorption of the Organ of Corti in Animals. M. H. Lurie, M.D., Boston 712

LXVII.—Roentgen Findings in Acute Infections of the Mastoid Cells. A. S. Macmillan, M.D., Boston 718

CONTENTS—Continued

PAGE

LXVIII.—Hearing in Chronic Otitis Media. Philip E. Meltzer, M.D., Boston 727

LXIX.—Massive Hemorrhage as an Otolaryngological Complication. Fred E. Motley, M.D., Charlotte, N. C. 737

LXX.—The Clinical Examination of the Hard of Hearing. Werner Mueller, M.D., Boston 756

LXXI.—Diagnosis and Treatment of Labyrinthitis Secondary to Tympanic Infection. Philip Mysel, M.D., Boston 761

LXXII.—Lateral Transthyroid Pharyngotomy. Henry Boylan Orton, M.D., Newark, N. J. 774

LXXIII.—Orbital Infections. Charles T. Porter, M.D., Boston 780

LXXIV.—Cavernous Hemangioma of the Nasopharynx. Julio Quevedo, M.D., Guatemala City 785

LXXV.—Endoscopy at the Crossroads. Lyman G. Richards, M.D., Boston 791

LXXVI.—Observations in Acute Otitis Media. John R. Richardson, M.D., Boston 804

LXXVII.—Surgical Treatment of Otosclerosis: Comparative Analysis of Results Using Different Technics. George E. Shambaugh, Jr., M.D., Chicago 817

LXXVIII.—Fourteen Years of Practical Experience With Sinus Problems in an Allergy Clinic. Francis L. Weille, M.D., Boston 830

The Scientific Papers of the American Broncho-Esophagological Association

LXXIX.—Adenoma (Mixed Tumor) of Bronchus: A Study of 35 Cases. Louis H. Clerf, M.D., and Carl J. Bucher, M.D., Philadelphia 836

LXXX.—Kodachrome Visualization of the Physiology and Pathology of the Tracheobronchial Tree. Paul H. Holinger, M.D., Chicago 851

LXXXI.—Fibrolipoma of the Laryngopharynx. F. W. Davison, M.D., Danville, Pa. 853

Society Proceedings

Sixty-fourth Annual Congress of the American Laryngological Association. Ludwig's Angina—Infections With Anaerobic Streptococci With Special Reference to Cranial Osteomyelitis 855

Obituaries 866

THIS VOLUME

OF THE

Annals of Otology, Rhinology and Laryngology

IS DEDICATED

TO

HARRIS PEYTON MOSHER

A.B., M.D., Sc.D., LL.D.

WALTER AUGUSTUS LE COMPTE PROFESSOR OF OTOLOGY, EMERITUS

AND

PROFESSOR OF LARYNGOLOGY, EMERITUS

OF

THE HARVARD MEDICAL SCHOOL

BOSTON, MASSACHUSETTS

THE CONTRIBUTIONS ARE BY HIS STUDENTS AND RESIDENTS, WHO HEREBY ACKNOWLEDGE THEIR INDEBTEDNESS TO HIM FOR HIS STIMULATION AND ENCOURAGEMENT IN THEIR PREPARATION FOR THE SPECIALTY OF OTOLARYNGOLOGY.

SEPTEMBER, 1942

DR. MOSHER Photographed at His Desk by Dr. Werner Mueller

ANNALS
OF
OTOLOGY, RHINOLOGY
AND
LARYNGOLOGY

VOL. 51 SEPTEMBER, 1942 No. 3

HARRIS PEYTON MOSHER

A member and past president of all the American otolaryngological societies; the president, from its organization, of the American Board of Otolaryngology; anatomist, sculptor, wood carver, artist, inventor, musician, teacher, trail blazer; a name associated with studies of the nasal sinuses, the esophagus, pus in the neck, brain abscess, the paraffin mould for skin grafting radical mastoid cavities, with many original instruments; dean of American otolaryngologists.

A descendant in the line of a sister of Paul Revere, Harris Peyton Mosher was born in Woodsford, Maine, a town named after his grandfather. His artistic ability was inherited from his mother, who was a musician and an artist; his inventive genius from his father, who was a civil engineer and an inventor.

His life fell into the Boston tradition: Harvard A.B. in 1892; M.D. in 1896; a surgical house officer of the Massachusetts General Hospital, to be followed by a residency at the Boston Lying-In-Hospital. During the Spanish-American War he served as contract surgeon at Montauk Point, Long Island. Then followed three years of general practice in Boston. During these years he became a member of the Department of Anatomy of the Harvard Medical School and here he developed his lifelong interest in anatomy.

In Berlin, he studied with Janssen and here his investigative mind turned to the anatomy of the nasal accessory sinuses. In Schwartze's old clinic in Halle he studied under Grünert. Returning to Harvard, he started a course on the anatomy of the accessory sinuses. His method of teaching by visualization, by drawing, by dissection, and by reduplication of the anatomical specimen in plas-

MEDICAL LIBRARY

ter, imprints on the memory facts that are never forgotten and his course became world famous. A Belgian student's description of it as "one of mud, glue, and plaster" fits, yet does not do it justice.

During World War I. Dr. Mosher volunteered early for military service and came out of the service late. After a tour of duty as a consultant in otolaryngology in both England and France, he was assigned to the personnel department of the Surgeon General's office. Here he was impressed by the need of better training in otolaryngology; and from this need, as a stimulus, the American Board of Otolaryngology was formed. Upon his retirement from the Medical Corps of the Army Dr. Mosher held the rank of Colonel.

World War II. finds him Chairman of the Section on Otolaryngology of the National Defense Council. He is kept busy as an air raid warden in Marblehead and with Mrs. Mosher is active in First Aid, the Red Cross, and British Relief.

In 1921 Dr. Mosher succeeded Dr. Coolidge as the Professor of Laryngology at Harvard and in 1933 he was, in addition, given the Chair of Otology, thus becoming the first since Oliver Wendell Holmes to fill two professorial chairs. With this appointment he became the Chief of Otology and Laryngology of the Massachusetts Eye and Ear Infirmary, one of the first full time otolaryngologists whose duties were entirely devoted to a medical school and to a hospital.

Dr. Mosher's contributions to medical knowledge are numerous. His studies on the ethmoid and its operative approach; on the esophagus, both clinical and histologic; brain abscess and the wire gauze drain; the paraffin mould for skin grafting radical mastoid cavities; on deafness caused by drugs; on osteomyelitis of the frontal bone; on chronic dacyrocystitis and an operative cure; on an operative approach to pus deep in the neck; and his many original instruments for esophageal and laryngeal surgery bear witness to his widespread interests.

Honored by all the American otolaryngological societies, he was the first American to deliver the Semon lecture and receive the Semon Medal in 1929. In 1934 he received the American Laryngological Association Medal. His honorary degrees are from the Universities of Pennsylvania, Colby, Wayne, and Jefferson. In 1939 he became Emeritus Professor at Harvard.

Dr. Mosher's influence has been widespread. To those who know him not, a porcupine quill exterior conceals a heart of gold. His

kindnesses, particularly to the younger otolaryngologists, have been many—yet concealed. Sham and hypocrisy excite his ire; sincerity and honesty his support. Devoted to his specialty, his medical school and his hospital he has enjoyed the sympathetic cooperation of Mrs. Mosher, who has supported him in all his struggles and who has rejoiced for him in his triumphs. Like the late George Apley, he has faithfully carried the torch of the Boston tradition. "Therefore, by their fruits, ye shall know them." St. Matthew 7-20.

L. A. S.

LII

LARYNGOCELE*

CHARLES H. ALLMAN, M.D.

AND

DAVID P. CORDRAY, M.D.

BOSTON

This paper will concern itself with a discussion and report of two cases of laryngocele.

Lindsay[1] described laryngocele as developing in three ways: "The most common type is said to be the internal laryngocele, a cystic dilatation appearing within the larynx. The second type is the superior external laryngocele, in which case the sac has perforated through the thyrohyoid membrane and appears as a swelling in the neck. The third type is a combined internal and external laryngocele."

He cited 35 cases as having been reported up to 1923 and additional cases since then, but the total number was still small. He reported a case of his own and by roentgenological examination with the planograph gave additional evidence regarding physiological action of the true and false cords and of the intratracheal pressures during phonation, muscular effort with the upper extremities, straining movements and swallowing.

In order to understand this condition clinically one must know something of the evolution and physiology of this particular portion of the larynx, namely the ventricle.

It is interesting that the larynx is primarily a valve, situated at the entrance of the pulmonary outgrowth and with the primitive function of preventing anything except air from entering the lungs. In the evolutionary processes certain modifications of this simple valve action became necessary and hence sphincter and dilator muscle groups have been added.

Because of the particular cases which we are to report, we must look more closely at the ventricle. Anatomically, the ventricle is a

*From the Department of Otolaryngology of the Massachusetts Eye and Ear Infirmary.

recess in the wall of the larynx bounded below by the true vocal cord which forms its floor. The outer wall and roof are formed by part of the aryepiglottic fold and in those animals so provided, the ventricle depends on the need for providing an inlet valve in a larynx which is of such a type (as in the human being) that without this modification there would be no possibility of efficient control of air entering or leaving the lungs.

The presence of a ventricular band is a later modification and is one found in an efficient form in only a few animals. In certain of the higher apes the ventricle becomes an actual pouch extending into the neck on either side and also superiorly and inferiorly. This, according to Negus,[2] acts to help in the fixation of the thoracic cage.

With this very brief background, let us go on to a consideration of the cases which we wish to report with the idea in mind that these were a definite atavistic reversion to our simian ancestors.

CASE REPORTS

CASE 1.—T.L.J., Canadian, aged 54 years, was admitted to the Massachusetts Eye and Ear Infirmary, May 10, 1939, complaining of hoarseness for the past five years. Along with the hoarseness he had observed a swelling in the left side of his neck, which could be reduced and made to disappear on swallowing or pressure directly over the mass.

For the past year he had a sensation of something in his throat. Occasionally he heard a peculiar noise when pressing on the mass with his hand. There had been no pain, loss of weight or hemoptysis, only slight discomfort when swallowing.

His family history was negative. He had had asthma some years previously, and typhoid fever, a gallbladder operation, and pneumonia twice.

Local examination showed a compressible mass about the size of a hen's egg on the left side of his neck, just above the thyroid cartilage. The left vocal cord and ventricle were chronically inflamed and thick grayish mucoid material was draining from the left ventricle. The patient was hoarse.

On May 11, 1939, operation was performed under local anesthesia. An incision was made along the anterior border of the sternocleidomastoid muscle on the left side. Another incision was made

Fig. 1.—Case 1. Anterior-posterior x-ray of the neck showing the unilateral air-containing laryngocele.

along the inferior border of the mandible, which joined the previous incision at its upper limit. The skin was reflected anteriorly exposing the sternocleidomastoid muscle and on the same plane and slightly anterior to it and just beneath the submaxillary gland the capsule of the cyst (laryngocele) was encountered.

Crossing over the cyst was an anomalous vein (the accessory jugular) which connects the common facial vein with the anterior jugular vein. This was retracted away and the capsule of the laryngocele dissected free.

The pedicle of the mass pierced the thyrohyoid membrane just above the upper rim of the thyroid cartilage and anterior to the superior cornu. It then progressed downward beneath the submucosa to empty into the ventricle of the larynx on the left side. The stalk was tied as close as possible to its exit into the ventricle and the mass removed intact.

Fig. 1 is an x-ray of the cyst and Fig. 2 a photomicrograph of pathology.

The patient was discharged home, eight days after operation with a normal voice. He was observed for two years, with no recurrence.

CASE 2.—J.D., an Armenian, 56 years of age, was first admitted to the Massachusetts Eye and Ear Infirmary in September, 1937, with a history of hoarseness for the past four months, morning cough, but no trouble swallowing. He also noticed a mass on each side of his neck which would disappear at times but reappeared occasionally when swallowing.

Examination of the larynx showed a mass on the epiglottis which protruded posteriorly into the larynx, obscuring the anterior portion of the vocal cords. This mass had the appearance of granulation tissue. There was a fullness of the left laryngeal wall which obscured the left vocal cord.

X-ray of the chest was negative and an x-ray of the neck region showed a mass on each side of the neck just above the larynx (Fig. 3).

Biopsy taken from the mass on the epiglottis showed chronic inflammation.

He was discharged for observation in the out-patient department and readmitted in November, 1937, and again in March, 1938, for biopsies. Pathological examinations of the biopsies demonstrated papilloma both times. He was discharged to the tumor clinic of the Massachusetts General Hospital, where he received x-ray therapy to the mass in his larynx and was followed there until November, 1938, at which time he was readmitted to the Massachusetts Eye and Ear Infirmary with marked difficulty in breathing and husky voice. Tracheotomy was done and the patient returned to the care of the tumor clinic.

He was admitted again in January, 1939, complaining of swelling in his neck, difficulty in swallowing and severe pain for three days. There was enormous swelling and redness over the entire neck anteriorly. An abscess had formed as a result of x-ray therapy. This was drained by a midline incision about the tracheotomy tube. He was discharged from the hospital and followed in the out-patient department until April, 1939, at which time he was admitted to the hospital for dissection of the laryngoceles.

Fig. 2.—Case 1. Photomicrograph of the wall of the laryngocele, showing it to be lined with stratified squamous epithelium and beneath this round-cell infiltration.

On April 21, 1939, operation was performed under local anesthesia, the incision being made along the anterior border of the sternocleidomastoid muscle. The cystic mass was encountered just anterior to and on the same plane as the jugular vein and the carotid artery, which were considerably more superficial than usual. The sac was dissected free of the surrounding fibrous tissue and its stalk followed and tied where it entered under the left wing of the hyoid bone. The sac was removed intact and was found to be filled with frothy fluid.

After this operation the wound healed partly but it was kept infected from the chronic draining abscess in his neck which had been opened and drained at one of the previous admissions. His general condition was poor and it was decided not to dissect the cyst from the other side of the his neck.

Fig. 3.—Case 2. X-ray of the neck, showing air-containing sacs on each side of the neck.

He was discharged to the Pondville State Hospital one month later from where we were unable to determine his subsequent course as he was reported to have left that institution against medical advice.

CONCLUSIONS

We have attempted to outline briefly the evolution and physiology of the ventricle of the larynx and to present two case histories with a description of the operative procedure employed to rectify this failure of adequate embryologic development.

520 COMMONWEALTH AVE.

REFERENCES

1. Lindsay, J. R.: Laryngocele Ventricularis, ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY 49:661-673 (Sept.), 1940.

2. Negus, V. E.: The Mechanism of the Larynx, St. Louis, 1930, The C. V. Mosby Co.

LIII

DEEP NECK INFECTION

August L. Beck, M.D.

New Rochelle, N. Y.

The chief purpose of this paper is to present a statistical report of an analysis of 100 cases of neck infection of all grades of severity. They have been classified into the following groups on the basis of applied anatomy and clinical manifestations: (1) pharyngomaxillary fossa infection, 48; (2) submaxillary region infection, 32; (3) visceral fascia infection (buccopharyngeal and pretracheal) 9; (4) prevertebral fascia infection (retropharyngeal), 3; (5) carotid sheath infection, 10; (6) cervical lymph gland abscesses, 24.

This grouping is somewhat arbitrary, but it does offer a means of classification which facilitates description. In many of these cases there was simultaneous infection in several of these regions; in fact it is rare for the infection to be confined to only one of these regions. For instance, infection was present in both the pharyngomaxillary and the submaxillary regions in 12 cases, and in eight of the cases of pharyngomaxillary infection there was also infection of the carotid sheath. Cervical gland abscess occurred six times in the cases of pharyngomaxillary infection, and 24 times in the entire series. Cervical adenitis, with or without abscess formation, occurred in 56 cases. The infection is apt to be predominant in the region where it first reaches the neck. Drainage of the locality of initial involvement is very important, and it is also important to obtain drainage in places of secondary involvements or extension, no matter how remote they may be, as in carotid sheath and mediastinum infections. In the 10 cases of infection along the carotid sheath, thrombosis of the internal jugular vein occurred three times. Osteomyelitis of the mandible occurred three times.

The largest number of cases (45) occurred between the ages of 2 and 12 years in children. The next largest group (32) was comprised of adults between the ages of 21 and 81 years of age. Between the ages of 1 month and 2 years, there were 11 cases; between the ages of 13 and 20 years, 12 cases. There were 45 females and 55 males.

Etiology is considered from the standpoints of bacteriology and portal of entrance.

Bacteriology is of increased importance since the sulfonamide drugs have become available for therapy. One or more of the varieties of organisms belonging to the pyogenic group are practically always present. Some form of streptococcus is most frequently found. The Streptococcus hemolyticus appears in pure culture more often than any other organism. It was present in pure culture in about 40 per cent of these cases. One or more forms of streptococcus appeared in over 80 per cent of all the infections. Many combinations of bacteria occur. The fusispirochete organisms of Vincent's angina are frequently found in the mouth, particularly when infection originates in the dental region. Alden[1] believes it is possible to abort such infections by the early administration of antiluetic arsenicals. In my experience the blood stream invader in such cases was the Streptococcus hemolyticus. It is possible that repeatedly negative blood culture reports may be explained by the possibility that the infecting organism may be an anaerobe. It is advisable that cultures should be grown under both aerobic and anaerobic conditions. No time should be lost in ascertaining the type of infecting organism, for one of the sulfonamide drugs should be administered as early as possible. The clinical course of cases is now capable of being tremendously influenced by the effects of the sulfonamide drugs. The earlier the drug is given the greater is its influence in terminating the infection. Even in the early stage the dosage should be such as to attain quickly a blood concentration of at least 10 mg. per 100 c.c. If such a concentration is attained while there is still an unbroken blood supply, the chances are good that there will be no abscess formation, and therefore no necessity for surgical drainage. Once an abscess has formed it will be necessary to drain it. Sulfonamides do not supplant drainage if an abscess has already formed. Usually, if after forty-eight hours there is no evidence of subsidence of the infection, it can be assumed that drainage is or will be necessary. During such a waiting period the administration of the drug should be assiduously continued. It is also reasonable to expect that even though the drug does not completely abort the infection in all of its phases, and that in one particular area there does occur a breakdown with abscess formation, such a localization is favorable for a drainage operation of comparatively minor character, and is a condition which is accompanied by only minor degrees of sepsis. After surgical drainage the administration of the drug should be continued at least until the patient is ambulatory unless conditions develop which contraindicate its con-

tinuance. Once the drug is discontinued it is more difficult to recapture control if there should occur a relapse.

Careful inquiry into the history is important because the visible evidence of inflammation at the point of entrance may have entirely disappeared by the time the infection manifests itself in the neck. A history of sore throat, from one to three or four weeks before the onset of symptoms, is sometimes elicited. Likewise, the patient may have had a tooth extracted a week or ten days before the onset of the neck infection, and the socket may have completely healed. I have repeatedly observed that in the early stage of a tonsil infection, while the inflammation is still limited strictly to the tonsil or a part of it, there may be no pain nor discomfort in the throat whatever; but as soon as the inflammation begins to involve the muscles of the tonsil bed, there is, immediately, pain and discomfort, and painful swallowing is promptly complained of by the patient. In such cases the onset may be announced only by systemic symptoms, such as high fever, malaise, headache, backache, nausea and even chills; in other words, toxic manifestations. The caustive condition in the throat might thus be entirely overlooked if it is not examined at the time. In such a case pain on swallowing, with gradual development of trismus, should arouse suspicion that a neck infection is in the making.

The infection may originate in the pharynx, tonsils, cervical vertebrae, nasal sinuses, temporal bone, dental region, salivary gland apparatus, esophagus and the respiratory passages. Trauma to the hypopharynx, esophagus, larynx and trachea is sometimes provocative of infections which extend into the neck. Fractures of the maxilla or mandible may also be followed by infection in the neck.

Knowledge of the portal of entrance makes it possible to anticipate the probable pathway of extension into the neck, and the probable place of localization of abscess.

In 50 cases the infection entered through the pharynx and tonsils. It is sometimes difficult to determine whether the infection enters through the tonsil or through the pharyngeal wall because when there is inflammation of the tonsil there is also present some inflammation of the pharynx, and vice versa. In seven cases the tonsils had been completely removed by surgery at a previous remote time. In six cases there were infected tonsil remnants. Infection followed tonsillectomy under general anesthesia in one case. In this series there was none after local anesthesia, though it is well known and appreciated that infection is more common after the use of local

anesthesia by injection. Infection followed removal of an adenoid under general anesthesia in one case. Infection followed inflammation in the nose and sinuses in four cases. In such cases there is often an accompanying pharyngitis, and a history of recurring attacks of sinusitis and pharyngitis. In these the sinusitis and pharyngitis may be with the patient long after the neck infection has been cured. Such was the history in two of these four cases. Trauma to the hypopharynx caused an abscess lateral to the hypopharynx and larynx in one case. Infection entered through the dental region in 19 cases, and in 11 of these it followed extraction of one or more teeth. Infection in the petrous part of the temporal bone caused pharyngomaxillary infection in one case. Otitis media and mastoiditis caused suppurative inflammation in the parotid gland in one case. Infection entered through the lingual tonsil in one case.

A deep neck infection resulting from inflammation of the pharynx and tonsil is most likely to be located in the pharyngomaxillary fossa. The carotid sheath is situated in the pharyngomaxillary fossa, and is therefore in close proximity, or in actual contact, with the infection, for which reason thrombosis and blood stream infection are strong possibilities. The superior constrictor muscle forms the inner boundary of the pharyngomaxillary fossa, and is covered by the buccopharyngeal fascia. Infection in the pharyngomaxillary fossa may therefore extend downward along this fascia, and cause a visceral, or pretracheal, fascia infection, and may thus produce a serious threat to the air passages in the form of edema of the larynx.

Pharyngomaxillary infection may extend downward into the neck along the carotid sheath, and, as pointed out by Mosher,[2] the sheath may be used as an avenue for the search of pus.

Sometimes small single abscess or multiple abscesses at or near the lower end of the tonsil may involve the visceral fascia directly, and gravitate downward along this fascia, and menace the air passages. Edema of the larynx is a frequent result of this type of infection. Pharyngomaxillary and visceral fascia infection may be present at the same time.

Pharyngomaxillary infection may extend into the parotid compartment, producing one of the triad of signs which characterize infection in the pharyngomaxillary fossa: (1) swelling of the lateral wall of the pharynx and fauces, (2) trismus and (3) external swelling over the parotid region.

Infection of the neck following infection from the cervical vertebrae first involves the prevertebral region. Eventually such an

infection penetrates the prevertebral fascia, and becomes either pharyngomaxillary or retropharyngeal. If it is lateral in location it becomes pharyngomaxillary, in which case it may extend downward along the recti capiti and the scaleni muscles. It is also in relation with the carotid sheath. If it is medial in location it becomes a retropharyngeal infection, and may extend downward along the longus colli muscles. In this case it would not be in close relation with the carotid sheath. If lateral, there would probably be torticollis or tilting of the head, toward that side. These infections are usually tuberculous and are analogous to psoas abscess in the lumbosacral region.

Lymph glands located in front of the body of the second cervical vertebra in the retropharyngeal region receive lymph drainage from the nasal sinuses, nasal chambers and nasopharynx. Infection in these parts, therefore, drains into these glands, and often causes abscess formation. When this happens there is produced the typical retropharyngeal abscess which occurs most frequently in infants and young children. This is classified as a prevertebral fascia infection. If seen before it has extended as low as the larynx, peroral incision is usually promptly curative. The head should be low, and suction at hand, so that when it is opened with a blunt-pointed instrument, such as closed scissors or artery clamp, the pus will not find its way into the deeper air passages. No anesthetic should be used. A mouth gag should be used for access and to minimize swallowing. If the swelling and inflammation have reached the level of the larynx there will be dyspnea, and in such cases it may not be possible to open the mouth for examination without provoking alarming dyspnea or asphyxia, usually from encroachment on the entrance to the larynx. In severe cases of this type it will be necessary first to restore the airway by tracheotomy, under local anesthesia, after which one may proceed as usual with the peroral incision through the posterior pharyngeal wall.

Infection along the prevertebral fascia may extend downward along the longus colli muscles into the posterior mediastinum. When the infection travels along the scaleni muscles it may reach the costocaracoid region or the axilla.

Infection from the temporal bone may point in one of several directions depending on which part of the temporal bone it involves. Infection in the mastoid process may point through the external cortex and produce an abscess which overlies the mastoid process in the posterior auricular region. This does not reach the neck except in proximity to the tip. Sometimes mastoiditis, manifesting itself

chiefly in the zygomatic cells of the temporal bone, is the source of infection in and around the roots of the zygoma, and the glenoid fossa, and is therefore in close relationship to the temporomandibular joint. Such an infection may extend into the zygomatic and pharyngomaxillary fossae if it erodes through the bone. Thorough eradication of the infection in the zygomatic cells, in the performance of the mastoidectomy, and drainage of the zygomatic fossa beneath the mandible and temporal muscle are the necessary surgical objectives. There was no case of this type in this series. When the infection breaks through the bone on the inner aspect of the tip in the region of the digastric groove there occurs what is known as Bezold's abscess. In this type of infection localization occurs below and anterior to the tip, and extends inward toward the pharynx and forward along the digastric muscle. The parotid region and upper part of the neck below the retromandibular region is the site of swelling and inflammation. It may extend as deeply as the pharyngomaxillary fossa, and as far forward as the submaxillary triangle. It is located beneath the upper part of the sternomastoid muscle, and may involve the carotid sheath.

Sometimes infection in the petrous apex of the temporal bone erodes through the inferior aspect of the bone, and involves the pharyngomaxillary fossa. Mosher[3] reported a case in which infection spread from the sphenoid, and perhaps from the posterior ethmoids, into the marrow spaces of the body of the sphenoid bone, and thence to the marrow spaces of the petrous apex from which, eroding through its inferior aspect, it spread to the pharyngomaxillary fossa. Drainage of the pharyngomaxillary space through the submaxillary fossa approach brought about subsidence of the suppuration in the regions of bone involvement, and complete cure of the infection.

Infection originating in the dental region of the mandible may extend into the neck by way of the labial, or buccal side, or by way of the lingual side, or by both. If the pathway of travel is along the labial side, it extends into the neck over the inferior margin of the jaw, and is superficial to the splitting layer of the deep fascia. In this type of infection incisions for drainage need not be placed more deeply than this superficial layer of the deep fascia unless there are lymph gland abscesses. There is no involvement of the tissues of the mouth floor.

If the pathway of travel is along the lingual side there is involvement of the soft parts of the floor of the mouth, base of the tongue and the region between the tongue and the internal pterygoid

muscle on the inner side of the body, angle and ramus of the jaw. It may also extend to the fauces, tonsil and lateral pharyngeal wall. This is the type of infection which, as soon as it extends below the mylohyoid muscle into the neck, becomes a Ludwig's angina. There is marked trismus, and usually a foul breath odor. The tongue becomes swollen and becomes crowded upward against the palate, and backward into the pharynx. The infection reaches the neck by direct penetration through the tissue spaces and lymph channels of the mylohyoid muscle, and by traveling along the submaxillary duct and salivary gland around the free part of the posterior border of the mylohyoid muscle. The lymphatic channels from the tongue, mouth floor and lower incisor region take a lateral direction just above the hyoid bone, and connect with the upper median deep cervical nodes on or near the carotid sheath.

Infection from the dental region of the maxilla may extend along the lingual side, and involve the hard and the soft palates. I have never seen neck infection by this pathway of travel. When the pathway of travel is along the labial side, the whole side of the face from the temporal region to the upper part of the neck, and from the nose and eyelids to the ear, is swollen. At the same time the inflammation extends deeply behind the maxilla, involving the sphenomaxillary, zygomatic and temporal fossae. It passes backward medial to the ramus of the jaw, and therefore will be found deep to the temporal muscle if it involves the temporal fossa. Drainage must go through the temporal muscle. The maxillary sinus is frequently involved.

Infection of the parotid and submaxillary salivary glands places infection in these particular compartments of the cervical fascia. It usually remains confined to these compartments, but infection may extend into the pharyngomaxillary fossa by way of the retromandibular process of the parotid gland. By involvement of the lymphatic glands in relation with these compartments the area of infection may be considerably enlarged. Infection in and around the submaxillary gland involves the submaxillary and adjacent upper cervical regions.

Infections in or along the esophagus extend along the visceral fascia, and are the most serious of all types of neck infection. Most frequently they follow trauma to the esophagus by foreign bodies or instrumentation. This type of infection is characterized by profound sepsis, and rapidly reaches the mediastinum along the visceral fascia, sometimes before there are visible signs of the infection in the neck. Dysphagia and odynophagia will be present, and also tenderness along the esophagus on deep palpation. This type of

infection requires the early placing of drains on the visceral fascia, and often, mediastinotomy. In external injuries of the neck the larynx and trachea may also be involved, and in these cases there may occur widespread emphysema of the neck. Crushing injuries, such as are sustained in automobile accidents, may cause fractures of the laryngeal or tracheal cartilages, with lacerations of the mucosa and submucosa, and perhaps cartilaginous collapse. In such cases mucosal and submucosal edema and swelling may seriously impair the airway, and even cause asphyxia, either immediately or as a later development. Visceral fascia infections are always serious. Lateral roentgenograms of the neck should be made to show the width of the shadow of the retropharyngeal tissues, and to show the presence or absence of air or gas in the retropharyngeal tissues.

Infection of the neck may follow injuries causing fractures of the maxilla and mandible. These are frequently compound, by lacerations through the alveolar process or maxillary sinus, and, because of this, infection of the wound is to be expected. Osteomyelitis is then also a possibility. War injuries in the region of the face and neck may be expected to increase the incidence of neck infection greatly. In these wounds which are characterized by extensive laceration, comminution, and actual loss of tissues, coupled with delays in the administration of surgical treatment, the establishment of infection cannot always be avoided. The value of the sulfonamide drugs in this connection has already been demonstrated. In addition to internal administration of the drug, local application in powder form or liquid spray may be used advantageously in some cases.

Usually complications are of a serious nature, but sometimes they are not serious enough to add greatly to the gravity of the condition. Unchecked neck infections may go on to fatal termination by one or more of three routes; namely (1) septicemia, (2) hemorrhage or (3) asphyxiation. Secondary involvement of any of the vital organs may be the direct cause of death. Examples of this are cerebral abscess, meningitis, pneumonia, nephritis, suppurative pancreatitis, hepatitis, etc. Rupture into the external auditory canal has been observed in two cases, thrombosis of the internal jugular vein in three cases, osteomyelitis of the mandible in three, bronchopneumonia in two, edema of the larynx in two, diabetes in two and a positive blood culture in four cases. The following have each been observed in separate cases: thrombosis of the sigmoid sinus, thrombosis of the cavernous sinus (with thrombosis of internal jugular vein), secondary hemorrhage from the pharyngomaxillary fossa

after operation, phlebitis of the internal jugular vein without thrombosis, inflammatory swelling of the calf of the leg with positive blood culture, paresis of the ninth, tenth and eleventh cranial nerves (simultaneous involvement), paresis of the twelfth cranial nerve, paresis of the pupillary fibers of the cervical sympathetic nerve, hip joint abscess, bilateral abscess of the calf of the leg, pelvic abscess, bilateral choked disc, cystitis, osteomyelitis of the maxilla, erysipelas, five days after recovery, secondary hemorrhage from veins of necrotic glands, lobar pneumonia, empyema, bronchopneumonia with scattered areas of consolidation, mastoiditis, acute suppurative otitis media, edema of the lungs, anterior and superior mediastinitis, acute glomerular nephritis (before advent of the sulfonamide drugs), scarlet fever, manifesting itself after drainage of cervical gland abscess, and abscess in zygomatic and temporal fossae.

There were three deaths in the series. In the first, the cause of death was septicemia, following thrombosis of the internal jugular vein and cavernous sinus, and failure to recognize the condition and to drain externally. In the second case the cause of death was mediastinitis and edema of the lungs, also with failure to drain externally. In the third case the cause of death was extensive bronchopneumonia with scattered areas of consolidation and inadequate external drainage.

421 Huguenot St.

REFERENCES

1. Alden, Arthur M.: Deep Neck Infection Secondary to Dental Disease, J. Am. Dent. A. 23:854-860 (May), 1936.

2. Mosher, Harris P.: President's Address, The Submaxillary Fossa Approach to Deep Pus in the Neck, Tr. Am. Acad. of Ophthalmol. & Otolaryng. p. 23, 1929.

3. Idem.: A Talk to House Officers and the Pharyngo-Maxillary Approach to the Tip of the Temporal Bone, Annals of Otology, Rhinology and Laryngology 49:3 (March), 1940.

LIV

ACUTE MASTOIDITIS: 1931-1941*

LAWRENCE R. BOIES, M.D.

MINNEAPOLIS

The decade just past has witnessed two events which must rank in importance with any previous milestones in the progress of otology. One was the development of our knowledge regarding suppuration within the petrous portion of the temporal bone. The other arrived with the application of sulfonamide therapy to acute middle ear and mastoid suppuration and its complications.

Approximately eleven years have passed since American otologists first became interested in petrositis. About five years ago chemotherapy entered the otologic picture.

This discussion is essentially an effort at "taking inventory" at this time on these two occurrences and has as a background a series of cases of acute surgical mastoiditis in 650 individuals during the years 1931 to 1941.

One hundred twenty-seven cases were private ones; 523 cases were done on the otologic service at the University Hospital (Minneapolis) mostly by members of our resident staff under supervision. It seems reasonable for the purpose of this study to group these cases together. The management of the cases was practically uniform. A majority of the public ward cases have been under my personal observation or supervision. A total of 697 mastoid operations were performed on these 650 patients. The extra number of operations refer to instances of bilateral mastoidectomy.

The first American contributions on petrositis appeared in 1930. In January of that year Eagleton presented a paper before the Eastern Section of the "Triological" Society entitled "Unlocking the Petrous Pyramid for Localized Bulbar (Pontile) Meningitis Secondary to Suppuration of the Petrous Apex". In June, Profant read a paper on "Gradenigo's Syndrome With a Consideration of Petrositis" at the meeting of the American Medical Association. In September, Friesner and Druss published their observations on "Osteitis of the

*Read at the Forty-eighth Annual Meeting of the American Laryngological, Rhinological and Otological Society, at Atlantic City, June 3, 1942.

Petrous pyramid" in the *Archives of Otolaryngology*. In this paper, they referred to a report of Mayer in the German literature in 1929 in which he reached the apex of the pyramid through a group of cells below the promontory and found an abscess which he drained. The patient made a complete recovery. (Other observations concerned with petrous suppuration had appeared in European literature many years previously. These included those of Bürckner in 1883, Muck in 1900, Goris in 1903, Gradenigo in 1905, and Baldenweck in 1908.)

In December, 1930, Kopetsky and Almour published in the ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY the first of a series of papers on "Suppuration of the Petrous Pyramid; Pathology, Symptomatology and Surgical Treatment", and advocated a special technic for draining the pyramid. This consisted first of a radical mastoidectomy, and then the introduction of a burr through the zygomatic root between the carotid artery, the cochlea and the surface of the pyramid.

During the next few years otologists became petrositis conscious. I have noted in the *Quarterly Cumulative Index* that in 1931 there were 21 contributions listed on the petrous pyramid. The number jumped to 37 in 1933, 62 in 1935, and 95 in 1937. Since then there has been a decline to 57 in 1939 and 28 in 1940.

From the accumulated experience of many observers, the following seem to be significant facts:

1. Suppuration within the pyramid containing pneumatic cells is not uncommon. It probably occurs as a concomitant of middle ear suppuration rather frequently, may be without significant symptoms, and heals when free drainage and resolution of the middle ear and mastoid suppuration occurs.

A number of studies on the amount of pneumatization of the temporal bone have been made. These estimate that the petrosa is pneumatized in from 11 to 35 per cent of cases. Fowler, Jr., and Swenson[1] suggest that these figures are misleading because they are based on different methods of observation and on mixed age groups. They state that in their collection of 369 adult temporal bones there is not a single one which does not have a cell or two somewhere in the petrous pyramid.

2. When petrositis occurs as a clinical entity, as evidenced by certain symptoms of significant pain, toxicity, discharge, etc., and becomes a potential special surgical problem, the term petrositis

alone is inadequate. More definitive localization, such as anterior or posterior petrositis or apicitis, becomes important.

The excellent correlations between the X-ray and pathologic findings reported by Fowler, Jr., and Swenson,[1] Lindsay,[2] and others, have improved our understanding of the location and frequency of the various sites and the X-ray projections which most accurately reveal these sites.

3. A majority of the cases of clinical petrositis can be adequately drained through cell tracts exposed in a thorough simple mastoidectomy. Radical mastoidectomy may be necessary for exposure and drainage in some cases. Localized suppuration in the apex of the pyramid which cannot be drained through the exposures of the simple or radical mastoidectomy requires one of the special technics such as have been proposed by Ramadier or Lempert to reach the apex safely and adequately.

Lempert has stressed the importance of considering the internal carotid artery as a guide to the exenteration of the apex, just as the sigmoid in its course through the temporal bone acts as a guide to a complete exenteration of the mastoid process.

In the 650 cases on which my observations have been made, a diagnosis of petrositis as a clinical entity was made 10 times. Among the 22 fatalities in the 650 cases (3.37 per cent) a diagnosis of petrositis was made twice at autopsy, once on a 4-month-old infant with a bilateral osteomyelitis of the pyramid, once in a child 6 years old. The temporal bones were not obtained for study in five more of the patients dying of meningitis. (Four of these had meningitis when admitted to the hospital.) This places the incidence of clinical petrositis as between 1.8 per cent and 2.6 per cent in this series of cases.

The 10 patients in whom a diagnosis of petrositis as a clinical entity was made had the pyramid drained and all of these recovered. Seven of the 10 patients were drained by exploring cell tracts found after thorough simple mastoidectomy. Two were drained by the Eagleton approach. One of the patients was transferred, after a radical mastoidectomy failed to locate a significant cell tract, to the Mayo Clinic where an Eagleton approach was used to drain an abscess of the apex.

It is my belief that the modern appreciation of the possibilities of a petrositis have improved the quality of mastoid surgery, particularly among the otologists who have been trained near to or

within the period in which this knowledge has developed. I also believe that, independently of the influence of the sulfonamides on the incidence of the complications of mastoiditis, we are to have fewer complications of the type that occur postoperatively, as a result of this improved surgery.

Sulfonamide therapy became important to the American otologist in 1937. Reports on the use of prontosil and sulfanilamide effecting cures of streptococcus meningitis complicating acute otitis media and mastoiditis appeared for the first time in American literature in that year. In the several years since, these first sulfonamides and their derivatives have had widespread clinical trial.

I have reviewed many of the reports on the value of the sulfonamides in the therapy of acute otitis media and mastoiditis. There have been only a few studies concerned with the role of chemotherapy in preventing otitis media.

The conclusions one obtains from a study of the literature may be stated as follows:

1. A majority of the otologists and a larger majority of the pediatricians believe that sulfonamide therapy has been responsible for a distinct decrease in the incidence of acute otitis media, and in the necessity for mastoid surgery in the mastoiditis complicating this otitis media.

2. Many of the studies lack controls. In those observations in which a control series has been used, there seems to be no evidence that the use of sulfonamide therapy has produced a marked change in the incidence of acute surgical mastoiditis due to its effect on acute otitis media.

The study by Williams and his collaborators[3] at the Mayo Clinic on 265 patients reports the use of sulfonamide therapy on 112 of these patients, with the other 153 used as controls. Among the 112 patients treated, there were 48 patients with infection caused by hemolytic streptococci. Six of these 48 developed toxic reaction to the drug so that the sulfonamide was discontinued. Nineteen of the 42 patients (45 per cent) who received adequate controlled therapy in the hospital required mastoidectomy. In 34 patients infected with hemolytic streptococci and not treated with sulfonamide therapy, 20 (59 per cent) came to mastoid surgery. Thus, an advantage of 17 per cent is seen in favor of patients adequately treated over the untreated group. A small group of patients with infection caused

by the Diplococcus pneumoniae showed a 21 per cent advantage in those receiving sulfonamide therapy.

A personal communication from Dr. Horace Williams, of Philadelphia, reveals a study in which a series of 200 cases were used; alternate patients received a sulfonamide. "There was no difference in the course of the otitis in those that had the sulfonamide. The sulfonamide was not given until after the discharge had begun. Since 1935, the number of mastoidectomies has materially decreased although routinely a sulfonamide has not been given except when we are comparing a series."

3. It is probable that the lessened incidence of acute surgical mastoiditis may be in a considerable degree influenced by a lack of severe epidemics of those upper respiratory tract inflammations which commonly influence the incidence and severity of otitis media.

4. It is probable, also, that sulfonamide therapy is promptly controlling some of these infections of which otitis media has been a complication.

The study of Sako, Dwan and Platou[4] reported complications in eight of 100 cases of scarlet fever treated with sulfanilamide. Five of these patients had otitis media, four of whom required mastoidectomy. In a control group of 100 cases not treated with sulfanilamide, complications developed in 41. Seventeen of these 41 had acute otitis media; four of the 17 required mastoidectomy.

In Fig. 1 I have compared some figures on the incidence of acute surgical mastoiditis from 1931 through 1941.

Curves I and II represent the figures reported by Ganz, Lyons, and Ferguson[5] from the Children's Hospital of Boston. For the year 1941, only the figures for the first six months of that year are available. The figure used for this 1941 point in the curve accordingly is based on that proportion. It, therefore, probably represents an approximation. In commenting on these figures, the authors stated that outpatients do not receive sulfonamides but that drug therapy has been used frequently in private cases. They believe that the drop from 1938 on in private cases has been due chiefly to the sulfonamides.

Curve III shows the incidence of acute surgical mastoiditis at the University of Minnesota Hospital. Curve IV shows the incidence of the same at a private hospital (Abbott) with the largest private pediatric service in Minneapolis. It is of interest to note how closely Curves III and IV parallel each other. A majority of the patients

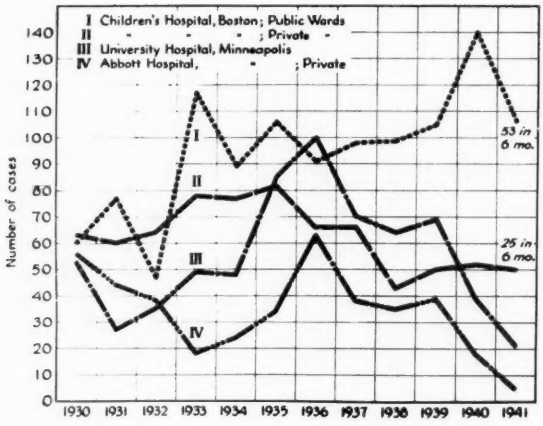


Fig. 1.

admitted to the University Hospital come from rural communities where they have had the customary home care of the rural physician who uses the sulfonamides generously.

The trend of these curves seems to imply that the use of sulfonamide therapy has produced a moderate decrease in the incidence of acute surgical mastoiditis in the patients receiving it at the Boston Children's Hospital. Most of the public ward cases and private cases from the Minneapolis hospitals received sulfonamide therapy and both groups show a parallel decrease. The high point in 1936 of these two curves of these Minneapolis hospitals was at the time of a measles epidemic. This year we have encountered another epidemic of measles and the indications are that if these curves are extended to include 1942, the trend will again be upward to show an increased incidence over 1941.

We are no doubt fully agreed on the usefulness of the sulfonamide compounds in the treatment of otitic sepsis and meningitis.

In 1930, I[6] reviewed the records of the Massachusetts Eye and Ear Infirmary and recorded a mortality of 25 per cent in cases diagnosed as lateral sinus thrombosis. Meltzer[7] in 1935 again studied the records of this institution and reported a mortality of 21.7 per cent in proven cases. In 1936, I[8] reviewed the records of 300 consecutive cases of acute surgical mastoiditis observed at the University

Hospital (Minneapolis) and in my private practice in a five-year period prior to March, 1936. There were 21 cases of well-established sepsis in this group with four deaths, a mortality of 19 per cent. These were all in cases occurring in the presulfonamide era. Since then (March 1, 1936, through the year 1941), I can record an additional 23 cases, 13 with positive cultures, 12 in whom the jugular vein was ligated. There was one death, giving a mortality of 4.4 per cent.

The one death occurred in a patient without a positive blood culture and in whom the jugular vein was ligated. The blood cultures were reported as beta-hemolytic streptococci in 12 of the cases and as gamma streptococci in one case. Most of the patients had sulfonamide therapy.

Our experience with otitic meningitis prior to the use of the sulfonamides was one of almost total failure. Today the picture is a different one. We can report our experience with meningitis within the sulfonamide era as follows: patients with positive spinal fluid, 10 (beta-hemolytic streptococci, 5; nonhemolytic streptococci, 3; pneumococci III, 2); patients without positive spinal fluids, 5; deaths, 3; mortality within these groups, 20 per cent; mortality in patients with positive spinal fluids, 30 per cent.

In all cases of otitic sepsis and of meningitis, we have felt it important to remove as thoroughly as possible the focus within the mastoid, doing a complete mastoidectomy and removing the lateral sinus plate and the middle fossa plate.

A consideration of the several sulfonamides, as to selection, dosage, toxic effects, etc., is not within the purpose of this discussion. Dosage has been well standardized, and the contraindications to the use of the drugs are well understood. At the present time, sulfadiazine is our drug of choice with infections due to beta-hemolytic streptococci. We prefer sulfanilamide first for infections due to the alpha-hemolytic streptococci (Streptococcus viridans), sulfapyridine for the pneumococcic infections, and sulfathiazol for staphylococcic infections except in meningitis where sufadiazine seems more effective. In my use of the sulfonamides, I have had the counsel of Dr. Wesley Spink.

Some otologists recommend the use of sulfonamide therapy for acute otitis media but not for acute mastoiditis. This raises the question: When does an acute mastoiditis begin? In any virulent case of suppurative otitis media, it is probable that a certain amount of inflammatory involvement of the mastoid structure occurs very

soon, probably within twenty-four to thirty-six hours after the suppurative process in the middle ear has become established. It now seems to be generally accepted that sulfonamide therapy is not as effective in inflammatory bone lesions as it is in soft tissue. The basis for its effectiveness is considered to be a matter of blood supply. If that is true, sulfonamide therapy in itself could not be expected to control effectively or heal with reasonable safety an area of inflammatory change "thrombosed off", or an area of abscess formation of any appreciable size within a mastoid structure. This has been proven in clinical experience in the past three or four years with the so-called "masking" effect of the sulfonamides on acute mastoiditis.

On the basis of these ideas in regard to action of the sulfonamides, it is my own practice to administer the drug at the very earliest moment in a case of acute otitis media in which there is evidence of any toxicity and to continue it while improvement is progressive. If, in a given case, there has not been improvement, and at the end of three or four days the drug has not controlled the process, I then prefer to interpret the needs of the particular case without the picture being confused by the drug. I also believe that early drainage of the middle ear is important. This means myringotomy unless adequate spontaneous drainage has occurred.

Some reports have recently appeared recommending the use of immune sera combined with chemotherapy.

SUMMARY

Two important developments in the decade just past have decreased the hazards of acute otitis media and mastoiditis.

The quality of mastoid surgery has been improved as a result of the development of our knowledge regarding suppuration in the temporal bone. This will decrease the incidence of postoperative complications.

The use of the sulfonamide drugs has decreased the incidence of acute otitis media and acute mastoiditis, particularly when the patient has had early and skilled medical care. It is reasonable to believe, however, that a share of the decreased incidence of these ear infections in the past two or three years is due to other causes, principally a lack of, or the better control of, the epidemic diseases which cause these infections.

The use of the sulfonamides has markedly lowered the mortality from otitic sepsis and otitic meningitis. An experience is reported

with 23 cases of otitic sepsis with a mortality rate of 4.4 per cent. This represents a 75 per cent improvement in fatalities from this cause. A mortality rate of 20 to 30 per cent in 15 cases of otitic meningitis is reported. This disease was formerly practically fatal.

APPENDIX

The fact that we have now accumulated nearly a five-year experience with sulfonamide therapy and more than a ten-year experience with the problems of petrositis interested me in obtaining a consensus of present-day opinion relative to some aspects of these two experiences. I recently sent out a brief questionnaire to 38 otologists whose writings, or association with a public ward or teaching service, or geographical location make their viewpoints of special value. Thirty of this number favored me with replies. The questionnaire contained these queries:

1. How much of a decreased incidence of acute surgical mastoiditis have you experienced in the past year or two (1940-1941)? The answers were unanimous in the matter of decrease, with estimates of from 25 to 95 per cent, particularly in private practice.

2. Has it been your experience that there has been a relative increase in the incidence of the complications of acute mastoiditis? Twenty felt not but eight had noted an increase in the complications.

3. Has there been in your experience a relative increase in the incidence of any one of the complications (petrositis, sinus thrombophlebitis, meningitis, brain abscess)? Several believe that there has been a relative increase in meningitis and sinus thrombophlebitis.

4. Do you generally recommend sulfonamide therapy in an ordinary case of acute otitis media? An acute mastoiditis? Or reserve it only for a complication of mastoiditis? Do you supplement with serum therapy? Nineteen used sulfonamide therapy in otitis media, 11 did not; 16 used it in mastoiditis, 14 did not; six used it in complications only; 22 used it in mastoid wound, seven did not. Eleven had used serum therapy in complicated cases, particularly in pneumococcic infections.

5. Do you place a sulfonamide powder in the mastoid wound at the completion of the operation? Which powder? Sulfathiazole was found to be irritating. A sulfonamide in the wound was felt to make little difference in healing if the mastoid was thoroughly done.

6. What has been your experience with the "masking" effect of the sulfonamides? Twenty-nine had experienced masking, three found it a serious problem. The majority considered the masking no particular problem.

7. Has it been your impression that the awakened interest in mastoid anatomy through the appreciation of the possibilities of a petrositis have bettered the quality of mastoid surgery in the past five years? Twenty-four found mastoid surgery improved, and six did not.

8. Is it your impression that there have been fewer postoperative complications as a result of this improved surgery? Eleven felt there had not been fewer postoperative complications while 19 felt there had.

1631 Medical Arts Bldg.

BIBLIOGRAPHY

1. Fowler, Edmund P., Jr., and Swenson, Paul C.: Petrositis—A Roentgenologic and Pathologic Correlation, Am. J. Roentgenol. 41:317-342 (March), 1939.

2. Lindsay, John R.: Petrous Pyramid of Temporal Bone: Pneumatization and Roentgenologic Appearance, Arch. Otolaryng. 31:231-235 (Feb.), 1940.

3. Williams, Henry L., Brown, Alex E., Herrell, Wallace E., and Ralph, Robert D.: Sulfonamide Therapy for Acute Otitis Media and Mastoiditis, Annals of Otology, Rhinology and Laryngology 50:336-347 (June), 1941.

4. Sako, Wallace, Dwan, P. F., and Platou, E. S.: Sulfanilamide and Serum in the Treatment and Prophylaxis of Scarlet Fever, J. A. M. A. 111:995-997 (Sept. 10), 1938.

5. Ganz, Robert N., Lyons, Champ, and Ferguson, Charles F.: Chemotherapy and Serotherapy of Acute Otitis Media, Annals of Otology, Rhinology and Laryngology 50:1185-1188 (Dec.), 1941.

6. Boies, L. R.: Lateral Sinus Thrombosis; Review of One Hundred and Eighty-Four Cases, Annals of Otology, Rhinology and Laryngology 41:227-237 (March), 1932.

7. Meltzer, P. E.: Treatment of Thrombosis of the Lateral Sinus, Arch. Otolaryng. 22:131-142 (Aug.), 1935.

8. Boies, L. R.: Extradural Inflammation; A Study of Its Occurrence in Acute Surgical Mastoiditis, Tr. Am. Acad. Ophth. and Otolaryng. 41:150-162, 1936.

LV

INTRASINAL APPLICATION OF SULFONAMIDE POWDER

LESTER A. BROWN, M.D.

ATLANTA, GA.

Two important changes in the treatment of purulent sinusitis have come about in the last few years: (a) There is less surgery. (b) The sulfonamide group of drugs has partially revolutionized the therapy.

The effect of a sulfonamide drug applied locally, whether in solution or as powder, is common knowledge. The solution is used widely as a sinus irrigation. In cases of purulent sinusitis of long standing which have been refractive to usual treatment, local and systemic, except radical operation, better effect seems to be obtained from local application of a sulfonamide powder, rather than a solution.

Sulfonamide powder may be applied to the mucous membrane of the sinuses by the intranasal route. Radical sinus operation is used now as a means of getting this powder into the sinuses, but the intranasal route seems to be just as effective.

Radical sinus operation is necessary in a limited number of cases, but in disturbing the mucous membrane of these infected sinuses, there is danger of spreading the infection. Especially is this true in the radical operation on the frontal sinus.

The treatment described below is applied to patients having pansinusitis of one or both sides, when the condition is an infection with pus and not an allergic state. There are five cases about which this article is written. These patients, with one exception, had histories of chronic purulent sinusitis of several years' duration. Some had had previous conservative treatment, but none had had surgery. They were not classified with the usual run of patients with purulent sinusitis, but were patients who would have been advised to have radical operation on the affected sinuses had it not been for improvement by this treatment. In fact, one of the patients previously had been advised to have radical operation on the frontal and maxillary sinuses. All five patients have been dismissed as well.

The following method is used: In the hospital, under local anesthesia (1) a submucous resection is done, unless the nasal septum is deviated sharply away from the infected side. (2) The anterior one-half of the middle turbinate is removed. (3) With a sphenoid punch forceps, the natural ostium of the antrum is enlarged to at least three times its normal size. (4) The nasofrontal duct is located, and the anterior ethmoid cells around this are entirely removed. (5) A rasp is introduced into the nasofrontal duct and the duct is enlarged so that a No. 3 eustachian catheter may be passed into the frontal sinus with ease. (It is necessary that the opening into the frontal sinus be large enough for the No. 3 catheter to fit loosely). No more ethmoid cells are removed unless the posterior cells or the sphenoid sinus is likewise infected. The frontal and maxillary sinuses are washed out with saline solution, through a No. 6F flexible ureteral catheter from a 10 c.c. syringe and 22 gauge needle, and this followed by injection of air from the syringe. Next, a metal eustachian catheter, usually a No. 2, is passed into the frontal sinus through the enlarged opening. A column of air from the wall pressure or small motor compressor, under extremely low pressure, is blown through the loosely fitting catheter. When it is ascertained that there is no obstruction to the return of the air from the sinus around the catheter (and this can be absolutely determined by the fit of the catheter in the opening to the frontal sinus, before injection of the air), a common powder blower, one-half filled with a sulfonamide powder, is inserted into the dilated end of the eustachian catheter and the air pressure attached to the powder blower. Sufficient powder is blown through the blower, through the catheter and into the sinus to cause some to come into the nose from the sinus. This procedure is repeated in the maxillary sinus through the enlarged natural ostium. Finally, a small amount of powder is sprayed directly from the powder blower into the anterior ethmoid region. Nasal packing of plain gauze is used only when a submucous resection has been done. This is a very light packing for the purpose of holding together the septal flaps, and it is removed in about eighteen hours.

Occasionally, a patient complains of severe pain during the fifth step of this procedure. This step is so important that should this occur, the patient is given a general anesthetic, however not until this part of the operation has been reached. Pentothal sodium, intravenously, has been the anesthetic of choice, because it acts rapidly, rarely causes a stage of excitement, and is administered entirely apart from the field of operation. A sterile airway, with catheter attached, is a part of the operating room setup, since anesthetists frequently

prefer to give oxygen while administering this anesthetic. Inasmuch as the mucous membrane of the nose has been anesthetized locally, there is no sneezing and very little bleeding. Irrigation of the sinuses with saline solution is omitted in this event, but the sinuses are insufflated with the sulfonamide powder.

The patient usually goes home on the third day, at which time the operated sinuses again are insufflated with the powder. Following this, the patient comes to the office twice weekly for some four to eight weeks for the insufflation. This procedure in the office is effected easily and without any discomfort to the patient. The nose is prepared by the use of a nasal spray of 1 per cent aqueous solution of cocaine hydrochloride which furnishes ample vasoconstriction and anesthesia. The sinuses are washed with saline solution before each treatment.

For the intrasinal insufflation, sulfanilamide powder, sulfathiazole powder, and a mixture of the two, have been used. Sulfanilamide powder has worked best. This may be due to its ease of injection and its greater solubility.

Two of the five cases are here reported in brief:

CASE 1.—A 50-year-old woman gave an account of purulent nasal discharge, with odor, from the right naris, of several years' duration. There were frequent attacks of pain above and below the right eye. Her present illness was an acute exacerbation due to a common cold. She spoke of having had pains and stiffness in many joints, especially in joints of the extremities. She stated that for over a year, on arising in the morning, it had been necessary to place her hands under warm running water before she could bend her fingers. When she awakened in the early hours of the morning because of cold, it was with the utmost difficulty that she could pull additional cover over herself. X-ray examination showed increased density of the right frontal and right maxillary sinuses with definite inflammatory reaction of the bone around these sinuses, indicating a right pansinusitis with pus, of long standing. Clinical examination revealed foul pus flowing from the right middle meatus, where also were several polypi. There were no polypi elsewhere in the nose, and no history nor evidence of allergy. She was treated in the office from Oct. 11, 1941, to Feb. 12, 1942, during which time the polypi were removed and the right maxillary sinus was irrigated with 5, 10 and 15 per cent aqueous solutions of sodium sulfathiazole at intervals of from one to five days. The patient felt improved during active treatment, but actual improvement of the sinusitis was only temporary. On Feb. 12, 1942, she was operated upon as described and dismissed from the hospital Feb. 15, 1942, following which she was treated in the office at three-day intervals. On March 10, 1942, there was no pus in any sinus. On April 15, 1942, she was dismissed as well. From the early part of March 1942, she had entirely recovered from any joint pains.

CASE 2.—A 36-year-old man first came into the office in December, 1940, with purulent left frontal and maxillary sinusitis. He told of having had purulent nasal discharge from the left naris for at least five years. During the past eight

weeks, there had been severe pain around and over the left eye, accompanied by an increased amount of purulent nasal discharge. On examination, the nasal septum was found to be so deviated to the left, that the left middle turbinate was obscured. A copious quantity of yellow pus flowed from this region, presumably from the left middle meatus. The left frontal and maxillary sinuses were completely opaque to transillumination. In January, 1941, a submucous resection was done and the anterior one-half of the left middle turbinate was removed. No sinus was entered. At this time it was seen that the pus actually did come from the left middle meatus. The patient was treated at intervals of from three days to six weeks throughout the entire year of 1941. Treatments consisted of left maxillary sinus irrigations with saline solution; enlarging the openings of the left frontal and left maxillary sinuses; left frontal and left maxillary sinus irrigations with 5, 10 and 15 per cent aqueous solutions of sodium sulfathiazole; sulfathiazole and sulfanilamide by mouth; aqueous solution of bismuth sodium tartrate, intramuscularly; extraction of a questionable left upper molar tooth. At times, the sinusitis seemed to improve, only to return to the original state when the interval between treatments was lengthened. The patient felt unable to take time off from work for sinus surgery and preferred the more conservative treatment. Finally, on Dec. 27, 1941, one year after the beginning of active treatment, the openings of the left frontal and left maxillary sinuses were again enlarged and this time the sinuses were insufflated with sulfanilamide powder after washing them clean of pus with saline solution. Following this, the patient was treated every seventh day. On Feb. 19, 1942, for the first time, there was no pus in the nose and the ostia of the frontal and maxillary sinuses and anterior ethmoid cells were open and free of any granulation. He was watched at less frequent intervals until March 10, 1942, when he was dismissed as well. He was last seen May 30, 1942, at which date he remained well subjectively and objectively.

The apparatus is part of any otolaryngologist's equipment. Simply a powder blower, three metal eustachian catheters, Nos. 1, 2 and 3, and air pressure.

CONCLUSIONS

Sulfonamide powder may be applied to the mucous membrane of the sinuses by the intranasal route.

The sulfonamide powder seems to be more effective than the solution in cases of chronic purulent sinusitis, probably because of its more lasting contact with the mucous membrane.

A treatment for those cases of chronic purulent sinusitis, which have not responded to the more common methods of treatment, is presented as another procedure to lessen the necessity for radical sinus operation.

478 PEACHTREE STREET, N. E.

The author wishes to express his appreciation to Dr. Homer Blincoe, Professor of Anatomy, Emory University School of Medicine, for his permission to use cadaver specimens for demonstration.

LVI

BILATERAL JUGULAR LIGATION FOLLOWING BILATERAL SUPPURATIVE MASTOIDITIS

MAURICE G. EVANS, M.D.

BOSTON

The internal jugular vein has often been ligated and resected for sigmoid sinus phlebitis or thrombosis complicating middle ear disease but actual ligation of both internal jugular veins during the course of bilateral suppurative mastoiditis is an exceedingly rare procedure.

To date there are only two such cases reported in the literature and to these the writer wishes to add a third with recovery.

REPORT OF A CASE

On Aug. 7, 1930, a boy, aged 8½ years, was admitted to the medical service of a local hospital complaining of loss of strength, pallor, fever and severe left frontal headache of one month's duration. The past history revealed, at 2 years of age, a right otitis media with spontaneous rupture and discharge for two weeks; mumps at 3 years of age and scarlet fever at 5 years of age. Four years previously his tonsils and adenoids had been removed because of frequent colds and one year prior to admission the child had had pneumonia. The family history revealed that three paternal uncles and one aunt had died of tuberculosis; and the child's mother was ill with a "leaking heart valve", a complication of scarlet fever during her childhood.

One month prior to admission to the hospital the child suddenly developed a sore throat and a head cold associated with fever and headache. The following afternoon he complained of pain in the right ear. This the father treated with warm irrigations and after two days the pain subsided. At no time was there any discharge from the ear. Since the onset of the present illness the child seemed generally listless, dazed, and was not very active. He frequently would fall asleep. His appetite was extremely poor, and every morning he would complain bitterly of left-sided headache. For the past three weeks there had been no ear complaints.

Physical examination on admission revealed a pale, thin, rather poorly nourished male child, lying apathetically in bed, hyperirritable and complaining of pains in the epigastrium. His skin was dry and hot and there was a very apparent loss of weight. The temperature was 103° and his pulse 120. The heart and lungs were clear, the eyegrounds and reflexes were normal. Both ear drums were equally injected and somewhat bulging. No mastoid signs were present. The W. B. C. was 9,300; R. B. C., 4,200,000; Hgb., 70 per cent, and a blood culture was negative. His urine showed a slightest possible trace of albumen. Two days later I first saw the child in consultation with the medical service, at which time the examination revealed a subacute nasopharyngitis with both ear drums fiery red throughout, bulging posteriorly and complete loss of landmarks. There was no mastoid swelling, tenderness or periosteal thickening. A bilateral paracentesis released serosanguinous fluid, culture of which showed hemolytic streptococci. X-rays were taken and the chest showed a chronic bronchitis; the sinuses, slight clouding of both antra; and the mastoids revealed slight clouding of the pneumatic structures in both sides; the cells, however, were not completely obscured.

During the next five days the temperature ranged from 98° to 104° with a pulse rate of 120. The child appeared extremely ill and complained bitterly of left frontal headache. The aural serosanguinous discharge had become purulent in character and the ear drums showed definite signs of resolution. Four days later, the mastoids were again X-rayed and reported as showing a definite increase in the clouding of the pneumatic structures on the left, while the appearance of the cells on the right had not changed much. The X-ray diagnosis was bilateral mastoiditis, worse on the left.

A lumbar puncture at this time showed a clear fluid with an initial pressure of 80, three cells, globulin 0 and normal sugar. The Tobey-Ayer jugular compression test was negative.

A bilateral simple mastoidectomy was performed completely uncovering both sigmoid sinuses throughout their mastoid course. The left mastoid was sclerosed and the antrum contained much granulation tissue which extended down along the posterior canal wall into a fairly large cavity filled with granulations and some pus. Because of the persistence of left-sided headache, the tegmen was removed and the middle fossa dura was exposed and appeared normal. The mastoid was then loosely packed with iodoform gauze and the upper portion sutured with silkworm gut. The right mastoid was then opened. The antrum contained a small amount of

granulation tissue, while the rest of the mastoid cavity was sclerosed and devoid of any pus or granulations. No dura was exposed but the sigmoid sinus was uncovered throughout its course and appeared perfectly normal. The cavity was packed in the same manner.

The patient, due to his poor condition, was then given a blood transfusion. He was cyanotic, his extremities were cold and his body covered with a cold perspiration. The temperature at this time was 100.2 , pulse 160 and respirations 26. For the next four days the patient seemed to improve somewhat. Cultures taken from both mastoids revealed hemolytic streptococci while repeated blood cultures were negative.

On the fifth postoperative day, the temperature again rose to 105 with a pulse rate of 120. There was profuse purulent drainage from both ears. The patient complained of feeling cold and of pain in both ears. Two days later, the temperature still was 105 with pulse 140 and respirations 30. The child continued to complain of hot spells followed by chilly sensations. Lumbar puncture and Tobey-Ayer tests were repeated and again were perfectly normal.

It was definitely felt that the patient's condition was due to a sigmoid sinus involvement but the question now arose as to which sinus was involved and which jugular should be ligated. Because of the persistent left frontoparietal headache, the X-ray findings, and the gross pathology found in the left mastoid at the time of the operation, I felt relatively certain that the left sigmoid sinus was the one involved and therefore decided to ligate the left internal jugular vein. This was done by placing two No. 2 chromic catgut ligatures about the vein and the neck wound was sutured. The mastoid wound was then reopened, the cavity cleaned of its granulation tissue, the sinus packed above and below in the usual manner and then incised. No clot was found and free bleeding was obtained from both ends. The cavity was then packed and the wound left open.

Following this procedure, the temperature, pulse and respiration remained normal for twenty-four hours for the first time. The patient seemed less restless and irritable but appeared quite sick. However, on the following day, the temperature again jumped to 103 and the pulse was 80 and irregular. His color was very poor, he complained bitterly of frontal headache, and was quite drowsy so that he could not be aroused for very long. Examination now revealed questionable Kernigs, slight stiffness of the neck, a typical meningeal cry, but the eyegrounds were still negative.

The following day the temperature was 102°, and the pulse, which was quite irregular, dropped to 60. The patient appeared extremely ill. Both fundi for the first time showed choking of the discs. A spinal tap revealed an initial pressure of 300 and a normal fluid, but the Tobey-Ayer test resulted in no rise on compression of the left jugular (the one that was ligated) and only a sluggish rise to 330 on compressing the right. There was slight edema of the left cheek just in front of the ear. That afternoon, under ether anesthesia, another spinal tap was done revealing a normal clear fluid with an initial pressure well over 600. The jugular compression test confirmed the previous findings of blockage of the right and left sinuses.

The right mastoid, which was by this time practically healed, was reopened and the cavity cleaned of its healthy appearing granulations. The sinus was packed above and below and then incised. An old firm grayish clot presented itself through the incision, but no bleeding was obtained. The wound was then temporarily packed and dressed and the right internal jugular vein was exposed in the neck. Two No. 2 chromic gut sutures were passed around the vein, firmly tied and the skin wound closed. Then returning to the right mastoid, the sinus was more extensively exposed, incised and many old clots removed, but free bleeding did not occur until the sinus was followed back by making a two inch counter-incision posteriorly through the skin and removing the overlying bone. When free bleeding was obtained from above and below, the sinus and mastoid cavity were packed with iodoform gauze, a firm pressure bandage was applied, and another blood transfusion was given. A culture of the sinus clot revealed hemolytic streptococci.

The following day the temperature was 99.6°, the pulse was 60 and irregular, the blood pressure was 85/55 and the eye discs showed more choking than on the previous day (3½ D). The patient was semiconscious, hard to arouse and complained of frontal headache. Another lumbar puncture revealed normal fluid with an initial pressure of 200. During the slow withdrawal of the fluid the child complained of terrific headache, therefore only 6 c.c. of fluid were withdrawn. Shortly after this procedure, the headache disappeared, the patient became much less irritable, felt much better and seemed to take an interest in his surroundings. From this time on, he made a rapid and uneventful recovery. He was discharged from the hospital on Oct. 10, 1930, both middle ears dry and both mastoid wounds healed well. His spinal fluid pressure remained about 500 and his discs showed 3½ D of choking.

For approximately four years he was followed in the medical, nerve and eye out-patient clinics. The choking of the discs persisted for about two years before it entirely disappeared. The vision in the right eye was 20/20 and in the left 20/200, but old school records proved that the child's vision was never better than 20/100 in the left eye.

In June, 1932, while riding a bicycle, the boy was struck by an automobile, causing him to fall and bump his forehead, and for some time he had spells of dizziness and headache but nothing unusual was found on examination. Lumbar puncture at this time revealed a normal fluid with an initial pressure of 150.

He was again seen by me in 1936 at which time his only complaint was blurring of vision in his left eye whenever he covered his right one. He informed me that he was doing well in school and at camp where he excelled in practically all sports including football, baseball, basketball, hockey and swimming.

A few weeks ago, eleven years after his operation, he consulted me in order to ascertain whether something could be done to improve the vision in his left eye, for he was anxious to join the maritime forces and go to sea, but was refused solely because of poor vision in his left eye.

COMMENT

The only ear complaint during the month prior to admission to the hospital was pain in the right ear which subsided after two days. Left frontoparietal headache was the most constant complaint during his entire illness. The first mastoid X-ray revealed bilateral mastoiditis and about one week later X-ray showed further progress of the disease on the left. At operation, much more pathology was found in the left mastoid than in the right, concurring with the X-ray findings. Repeated Tobey-Ayer tests prior to the ligation of the left internal jugular vein were persistently negative. Taking all these factors into consideration and, in spite of the right otalgia at the onset of the illness, the writer felt relatively certain that the left sigmoid sinus was involved and hence ligated the left internal jugular vein.

The patient however, showed no improvement and the examination next day revealed for the first time choking of the discs, edema of the left cheek, a high initial spinal fluid pressure and obstruction to both lateral sinuses as evidenced by the positive Tobey-Ayer tests.

These findings together with the old grayish clot found later in the right sigmoid sinus indicated that this sinus was thrombosed even before the left jugular vein was ligated.

It is interesting to note that, in spite of the persistence of marked papilledema for almost two years after the double jugular ligation, the patient's vision returned to its preoperative state.

REVIEW OF THE LITERATURE

Seven cases of bilateral internal jugular ligation (not of otitic origin) were found in the literature reported by general surgeons.

In 1900 Ewald[1] resected both jugular veins during an extensive neck operation for malignant glands. This patient died before regaining consciousness.

In 1902, Baldwin[2] reported a case of synchronous ligation of both jugular veins while removing large tuberculous masses from both sides of the neck. Both these veins were opened accidentally and required ligation. The patient made an uneventful recovery and was discharged from the hospital two weeks later without any evidence of circulatory disturbance save for a slight puffiness of the face.

Dangel,[3] in 1905, simultaneously resected both internal jugular veins during the removal of malignant cervical glands. This patient had slight cyanosis of the face lasting only a few seconds, and also made an uneventful recovery.

In 1909, Duval[4] reported three cases of bilateral jugular ligation. The first was that of a 47-year-old man with epithelioma of the chin, floor of the mouth and the lower jaw. Immediately following the ligation of the second jugular vein the patient's face became not only violet but black. This discoloration gradually extended down to the neck and soon the respirations ceased. Suddenly the patient had a profuse hemorrhage from the mouth and throat, the blood being absolutely black. After this spontaneous venesection, the patient began to breathe again and his face became violet and then pink. It was then possible to complete the operation. Except for a slight facial violet tint lasting for two hours, together with considerable parotid swelling for a day, the postoperative course was uneventful. Thirteen days later, the lower maxilla, the chin tissues and a part of the floor of the mouth were resected and the patient then made a good recovery.

The second case was a resection of the left internal jugular vein which was invaded by cancer. Sixteen days later the same oper-

ation was performed on the right side and while the wound was being sutured the patient became cyanotic and stated he did not feel well. His face soon became darker in color and swollen and his eyes became injected with blood and protruded. He died in about one minute.

In his third case, Duval waited one month between the ligations of the two jugulars. First he removed the entire left internal jugular vein because of epitheliomatous involvement. Convalescence was uneventful, and one month later the right internal jugular vein was ligated slowly. The patient's face became cyanosed and gradually his respirations became rapid and shallow and he died suddenly on the third postoperative day.

Le Grand[5] in 1922 also reported a case, but I have been unable to obtain a copy of his original report.

The literature also revealed eight cases of bilateral involvement of the sigmoid sinuses secondary to bilateral suppurative mastoiditis. Of these, six had both sigmoid sinuses incised and packed and only one jugular ligated, while in only two cases were both the internal jugular veins actually ligated or resected.

Gruening[6] in 1905 reported a case of bilateral simple mastoidectomy in a woman 22 years of age. High fever, chills, nausea, vomiting and choked discs followed, and twenty-three days later the left sigmoid sinus was incised and a large thrombus was removed, followed by free bleeding from above and below. Because of tenderness along the right jugular vein, the right jugular was ligated, the right sigmoid sinus incised and a large clot was removed, five days later. After a stormy convalescence which necessitated a second ligation of the right jugular vein at its entrance to the subclavian, the patient recovered. The only sign of intracranial circulatory disturbance was choking of the discs which eventually subsided with resultant good vision.

In 1915, Smith,[7] following a bilateral mastoidectomy, incised and packed both sigmoid sinuses. From the left sinus, there was good and free bleeding, while from the right only slight bleeding was obtained. Some time later the right jugular vein was ligated and resected and the patient recovered. No untoward symptoms were recorded.

Stickney,[8] in 1919, reported a case of bilateral mastoidectomy followed by chills, fever and joint complications. Incising both sigmoid sinuses, he obtained sluggish bleeding only from the right one and proceeded to ligate and resect the right internal jugular

vein. This was followed by complete recovery without any signs of increased intracranial pressure except for slight twitching of the left arm which was present for only a few hours.

The fourth case was reported by Tobey[9] in 1923, that of a patient with a persistent low-grade infection of both mastoids associated with signs and symptoms of sinus phlebitis. The right sigmoid sinus was incised and the right jugular was tied, but the patient became progressively more toxic. Six days later the left lateral sinus was incised and because free bleeding was obtained from both ends, the sinus was packed and the jugular vein was not ligated. Following this procedure, the patient made an uneventful recovery. At no time, either before or after the sinus operations, did the patient show any signs of increased intracranial pressure or circulatory disturbance.

Schlegel[10] in 1906 and Dausend[11] in 1910 each reported a somewhat similar case.

The first case of actual ligation and resection of both internal jugular veins was reported by Hastings[12] in 1926. Following a bilateral mastoidectomy the right sigmoid sinus was incised and found to be thrombosed down to the facial vein. The right internal jugular vein was ligated and one inch was resected. However, the patient not only failed to improve but the previously noted choking of the discs became progressively worse. Seventeen days later incision of the left lateral sinus revealed definite thrombosis, therefore the left internal jugular vein was tied and a portion resected. This was followed not only by a quick recovery, but the patient's vision gradually returned to normal.

Brown,[13] in 1941, reported the second such case. A 5-year-old male developed double mastoiditis following measles and bronchopneumonia. Both mastoids were exenterated but the patient remained febrile and later fluid was withdrawn from a painful right knee. However, septic temperature persisted and the mastoids were explored a second time. At this time, granulations were found on the left sigmoid sinus and a few days later the sigmoid sinus was opened. No thrombus was found, so the sinus was packed and the left internal jugular vein ligated above the facial. The daily high temperature subsided, but ten days later another chill occurred associated with a rise of temperature to 105°. Repeated positive blood cultures were obtained. Consequently five days later, the right sigmoid sinus was investigated and no thrombus was found. The sinus was packed, and the right internal jugular vein was also ligated above the facial. At the end of a week the temperature was normal

and blood cultures were negative. There was a moderate amount of dilation of the temporal, facial and external jugular veins together with increased tortuosity of the retinal veins which lasted for two weeks, and five weeks after the second ligation the patient was discharged well from the hospital.

DISCUSSION

Eminent authorities in anatomy and physiology have in the past consistently believed that ligation of both internal jugular veins would necessarily be a fatal procedure. Chamberlain, in discussing Tobey's[9] paper, speaks of a rapidly fulminating case of double mastoid involvement with symptoms of lateral sinus thrombosis. The question of double ligation of the jugulars was discussed with heads of the Departments of Anatomy and Physiology. Both professors advised against operative interference and the case went on to a fatal termination.

Hastings[12] in his paper also speaks of one of his confrères who was confronted with the problem of bilateral jugular thrombosis and ligation, but was strongly advised against such a procedure by the professor of anatomy at his university. This patient also died without resection and no necropsy was obtained.

Other writers have contended that marked intracranial circulatory disturbances would necessarily result from obstruction of the normal cranial venous return. Klestadt[14] states that when an anomaly of a lateral sinus exists (usually the left) or when the superior longitudinal sinus passes entirely to the right without any anastomosis at the torcular, the patient is likely to develop serious intracranial complications or may even die if the other vein becomes occluded or is ligated. This is more particularly true in cases where both internal jugulars may have to be ligated.

Precechtel[15] of Prague, in 1934, discusses disturbances in blood circulation within the cranial cavity, and states that when impairment of normal arrangement of the venous channels occurs as in ligated jugular veins, various intracranial symptoms may be noted; e.g., impaired consciousness increasing to unconsciousness, mania, apathy and amentia, headache, vomiting, retarded pulse, choked disc, affected sight ranging to blindness, convulsions, paresis of limbs, labyrinth irritation, disturbed heat regulation, glycosuria, polyphagia and obesity, aphasia and venostasis in the orbit and in the face.

It is interesting to note that in all the cases reviewed in this paper, temporary papilledema and a variable amount of venostasis

of the head and neck were practically the only complications observed. It is also interesting to note that in the 16 patients with impaired cranial venous return only three died, 12 completely recovered and in one the result is unknown.

Therefore, it is reasonable to conclude that bilateral internal jugular ligation is not necessarily a fatal procedure, nor is recovery necessarily associated with any untoward permanent symptoms of impaired intracranial circulation.

SUMMARY

1. A case of ligation of both internal jugular veins with complete recovery following acute bilateral suppurative mastoiditis is reported here in detail.

2. A comprehensive review of the literature on this subject is presented, including: (a) Seven cases of bilateral jugular ligation (not of otitic origin) reported by general surgeons of which three recovered, three died and the end result in one case is unknown. (b) Six cases reported by otologists where only one jugular was tied but both sigmoid sinuses were obliterated by incision and packing. All patients recovered with practically no serious signs of circulatory disturbance. (c) The only two cases of actual ligation of both internal jugular veins, fully recovered without any residual signs of increased intracranial pressure or circulatory disturbance.

3. The postoperative complications of bilateral internal jugular ligation are discussed.

CONCLUSIONS

It must be obvious from the literature and from the case here reported that there is no firm basis for the fear and trepidation of ligating both internal jugular veins should such a necessity arise. The procedure is not necessarily fatal, nor do intracranial symptoms necessarily occur when the normal venous return from the cranial cavity is materially influenced by this operation, for sufficient collateral circulation is soon established.

416 MARLBOROUGH ST.

BIBLIOGRAPHY

1. Ewald, C.: Tod unter vasomotorishen Störungen nach Resection beider Venae jugulares internal, Wien. Klin. Rundschau. 14:673, 1900.

2. Baldwin, J. F.: Synchronous Ligation of Both Internal Jugulars, Ann. Surg. 36:394, 1902.

3. Dangel, Max: Ueber die Unterbindung den Vena jugularis interna. Ein Fall von doppelseitiger Unterbindung, Beitr. z. Klin. Chir. 16:495, 1905.

4. Duval: Ligature des deux jugulaires internes, Bull. et mem. Soc. de chir. de Paris 35:224, 1909.

5. Le Grand: Un cas de ligature bilateral de jugulaires internes, Normandie méd. 33:58, 1922.

6. Gruening, E.: Case of Bilateral Thrombosis of Sigmoid Sinus With Recovery, Tr. Am. Otol. Soc. 9, 10:518-520, 1905-1907.

7. Smith, C. H.: Double Mastoid With Septic Sinus Thrombosis, New York M. J. 101:407, 1915.

8. Stickney, O. D.: Bilateral Acute Suppurative Otitis Media With Symptoms of Sinus Thrombosis, Laryngoscope 29:90, 1919.

9. Tobey, H. G.: Double Acute Suppurative Mastoiditis With Symptoms of Sinus Phlebitis or Thrombosis, Tr. Am. Laryng., Rhinol. and Otol. Soc. 29:6, 1923.

10. Schlegel, G.: Ein Fall von doppelseitiger Sinus thrombose, mit ein seitige jugularis Unterbindung, Arch. f. Ohrenh. 69:176, 1906.

11. Dausend, J. A.: Ein Fall doppelseitiger Sinus thrombose, Beitr. z. Anat. Physiol., Path. u. Therap. d. Ohres. 4:394, 1910-1911.

12. Hastings, Hill: Bilateral Jugular Resection for Bilateral Sigmoid Sinus Thrombosis (Otitic), Arch. Otolaryng. 4:58, 1926.

13. Brown, J. MacKenzie: Bilateral Jugular Ligation With Recovery, ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY 50:280 (March), 1941.

14. Klestadt, Walter: Zerebrale Symptomenkomplexe bei otogener Sinusphlebitis, Ztschr. f. Laryngol. 13:83 (Nov.), 1924.

15. Precechtel, A.: Contribution to the Question of Disturbances in Blood Circulation Within the Cranial Cavity in Cases of Thrombosed Sigmoid Sinus and of Ligatured Jugular Vein, Psychiat. en neurol. 38:526-533 (May-Aug.), 1934.

LVII

THE SIGNIFICANCE OF THE NOSE IN ALLERGIC STATES

RALPH J. FRACKELTON, M.D.

LAKEWOOD, OHIO

The purpose of this paper is to consider the nose and accessory sinuses in their relation to that specific reaction called allergy, and to discuss certain phases of treatment.

Because of its location, the nose has become highly adaptive to environmental changes. It becomes at once the portal of entry for foreign material and the battleground for the mechanisms of defense, whereafter the resulting products are disseminated to other parts of the body. For this and other reasons its relation to allergy and immunity is somewhat unique.

Rich[1] has described immunity and allergy as follows: "When a foreign protein, whether of bacterial or non-bacterial origin, finds its way undigested into the tissues, certain changes usually take place in the body as a result of contact with that protein. The exact nature of these changes is still obscure, but the effects which they may produce if the same protein reaches the tissues again are familiar in the form of various 'hypersensitivity reactions' to foreign protein. Depending upon conditions the reactions may appear in the form of anaphylactic shock, asthma, hay fever, serum sickness, 'food allergy,' the Arthus phenomenon, or the tuberculin and other similar reactions to the proteins of bacteria. . . . We are still far from understanding properly the precise influence of hypersensitivity in most infections, but in an ever-increasing number of diseases it is becoming evident that the various lesions and symptoms which were formerly assumed to be the direct result of specific bacterial poisons are actually the results, not of bacterial poisons, but of the hypersensitive state and there is, therefore, at present a widespread interest in the significance of this remarkable condition."

Antiallergic treatment has been an aid to the determination of which diseases are allergic in nature. As far as the nose is concerned, mucosal tests with various foreign proteins have produced a great variety of symptoms. A most imposing list has been compiled by

Kuhn and Linton.[2] They found that mucosal testing brings about "edema of the turbinates, rhinorrhea, sneezing, itching of the nose, itching of the roof of the mouth, headaches (usually unilateral), blurring of vision, pain in the eyes, scintillating scotoma, dizziness, vertigo, some loss of hearing (mostly subjective), tinnitus, earache, pain in the mastoid region, pain and fullness in the sinuses, Sluder's syndrome, neurotic symptoms (mild and irrational), rheumatic pains, pharyngitis, edema in the throat, coughing, asthma, indigestion, nausea, irregular menstruation, uterine pains, edema of the gums, dyspnea, increase in saliva, muscular weakness, hypertension, drowsiness, backache, fever, hot flashes, hives (usually on the side of positive reaction), and leucopenia, which will check closely with the leucopenia produced by ingestion." If these various symptoms, some mild and some alarming, may immediately follow the single application of allergens to the allergic nasal mucosa, it requires little imagination to envision the degenerative effect upon the circulatory, endocrine, and nervous systems, as well as on internal organs of repeated nasal absorption of specific bacterial and nonbacterial proteins. As Rich[1] states: "In a very realistic sense it may be said that the presence of hypersensitivity converts, in effect, a bacterium which does not produce a toxin into one that does, for it converts harmless products of bacterial disintegration into powerful and even lethal poisons." Similarly we see the soothing lycopodium become a powerful irritant to the nose and such excellent foods as milk and cereal produce local and remote allergic manifestations.

It is well to consider clinically the allergic reaction as different and distinct from that of the antibody formation of antibacterial immunity. Although an allergic state may accompany certain infections and although some allergic individuals react to most infections in an allergic manner, the subject is clarified when allergic and immune reactions are considered as separate entities. This distinctiveness is emphasized in those patients whose diverse infections over a long period of time are never accompanied by any known allergic reaction.

When one of the tissues of the nose is invaded by infection, the cells elaborate antibodies, which thereafter destroy or counteract the pathogenic effect of the organisms. To stimulate an increase of antibodies, vaccines made from similar organisms are employed, so that at the time of reinfection there will be excessive amounts of antibodies available. Rich[1] points out that "allergic inflammation, developing about bacteria after they reach the tissues, is, in the absence of specific immunity, incapable of preventing the spread of bacteria

or protecting against the infection." Tissue, in an allergic state, is conceded often to be more susceptible to infection unless it is accompanied by a degree of immunity independent of the allergic state.

Now the state of hypersensitivity appears to have points of similarity to immunity. First, it is a reaction to the presence of foreign protein. Second, the reaction results in the formation of new substances and an increase in the activity of the phagocytic cells. In immunity the cells of the reticuloendothelial system probably elaborate lysins, precipitins, agglutinins, etc., to destroy infection, and the natural phagocytic qualities of the cells may be thereby enhanced.

Although the entire body is subject to bacterial invasion, the nose and gastrointestinal tract are the potential points of entry for undigested proteins. From postnasal dripping the gastrointestinal and respiratory tracts receive the secondary products of bacterial and inhalant reactions present in the nose.

Allergy and immunity reactions do not necessarily run along parallel lines. They may exist together or independent of each other. Whereas the immunity reactions by antibody formation against bacterial proteins are desirable in the host, the allergic manifestations resulting from the combining of the allergen with its specific reagin are highly undesirable under certain conditions, whether it be in inhalant, food, or bacterial protein. Vaccines containing bacterial antigens are employed in increasing doses to raise the immunity by stimulating antibody formation. On the other hand, we also use increasing doses of specific allergens to induce a refractory (nonreacting) state in the host whereby allergic manifestations are avoided. The hypersensitivity in the host generally outlives the refractory period thus induced. Symptoms may then recur on renewed exposure and a new series of allergen injections be required to set up a new refractory state, improperly called "desensitization". Repeated and prolonged refractory periods may possibly favor recovery from hypersensitivity to a certain extent.

In the nose every allergic manifestation that is accompanied by sufficient mucosal swelling is accompanied by a new inoculation with the undigested foreign protein. If the nasal allergic edema is kept in abeyance over a long enough period, the host will be relieved of new inoculations incident to the allergic reaction. As stimulation lessens, hypersensitivity may tend to subside. The allergic regimen reaches its greatest practical heights when it concerns itself with withdrawals of the offending allergens of foods, inhalants, physical

agents, and bacteria, and at the same time raises the antibacterial immunity by the use of stock and autogenous vaccines, vitamins, endocrines, and nutritional factors. So-called "desensitization" by allergen administration is merely an adjunct to the above treatment.

In order to understand how the nasal inoculations (or infiltrations) may be prevented, let us consider normal nasal filtration so carefully studied by Proetz.[3] In the nose the same situations which favor serious irritation by allergens also favor invasion by bacteria. Foreign particles of all kinds which pass the vibrissae of the nasal vestibule are likely to fall upon the backward moving cilia-propelled sheet of mucus. The natural air currents of the nose and the difference in the electrical potential of the nose and particle attract the particle to the nasal wall. There is also reflex narrowing of the olfactory slit by the filling of venous lakes in the erectile tissue of the turbinates. After a moist turbinate has lightly contacted the nasal septum, its retraction leaves across the nasal chamber a series of bridging threads of delicately spun mucus.

The nose secretes a large amount of moisture, which has been estimated to be between twelve ounces and two quarts in twenty-four hours, with variations dependent upon such factors as air humidity, water intake, temperature, etc. The epithelial lining is admirably adapted to osmotic changes. Hansel[4] states: "The vasomotor system of the nose is considered as a complex and unstable mechanism which involves a balance between arteriomotor, venomotor, and capillomotor nerve fibers and chemical substances exerting regulatory influences." The rapidity of absorption is influenced by the integrity and rate of ciliary propulsion of the mucous sheet, the thickness of the basement membrane and the state of capillaries and lymph channels in the highly vascular tunica propria. The evidence of its capacity to absorb is noted after topical applications of internal secretions introduced on cotton into the nose. Pituitrin, thus absorbed, soon induces uterine contractions during pregnancy. Toxic substances, such as cocaine, may be quickly absorbed and cause immediate circulatory collapse. Odors are also immediately absorbed for speedy perception by the olfactory nerves.

If a foreign substance reaches the nose in an allergic individual, the resulting manifestations of allergy cause a serous exudate from the mucosa, which in turn becomes swollen and boggy and assumes a bluish pallor. The liberation of histamine or histamine-like substances favors the engorgement of capillaries and lymph channels and reduces cellular oxidation and nutrition.

Fig. 1.—Position of hand for continuous postnasal irrigation, using two quarts of isotonic solution of sodium chloride (108°) brought to three-way adapter by gravity from container placed about five feet higher than head.

The soft nasal epithelium often becomes swollen to the limits of its expansion within its bony walls. The resulting pressure is known to have a profound reflex effect upon the vascular and nervous systems. (Fig. 3.) Between the turbinates, the lateral nasal wall and the septum, the offending particles are caught into the stagnant sheet of mucus, pressed against the flattened epithelium at pressure points and thus absorbed undigested into the epithelium. By phagocytosis they are carried to the tunica propria to constitute an allergic inoculation for the perpetuation of hypersensitivity to those specific foreign substances. This injection into the blood and lymphatic systems of relatively large amounts of antigen in infection and the injection of allergen in allergic individuals account for the sudden onset of the symptoms of nasal infection or nasal allergy. When both reactions occur simultaneously, as they are almost bound to do in the

course of time, the resulting episodes are often extremely alarming, but seldom fatal.

When septal spurs, marked deviations of the nasal septum and hypertrophies of turbinates are asymptomatic, they may be disregarded, but in allergic states they must be seriously considered as interfering with the defensive mechanisms of the nose, for they may favor mucosal ulceration at the times of congestion by their continual or easily induced compressions along contact points.

The removal of a septal spur has been known to be effective in reducing or eliminating the inoculation of the allergen as also is the irrigation of a dust-laden antrum. The spur might be said to be "rubbing it in," while the antrum acts in the capacity of a reservoir. A recently examined patient suffered severe pain and burning sensation in one jaw and cheek with no evidence of inflammation. The antrum appeared clear by transillumination, but an irrigation brought out a very small amount of foamy white mucus flaked with dust particles and was followed by immediate relief. This represents the irritation of a reservoir in its simplest form. The accessory sinuses have a capacity of about 37 c.c. and one small ethmoidal cell, well concealed beneath or in the middle turbinate, may hold sufficient foreign material to keep up an inflammatory process in the nose for years. During that time the mucoid secretion changes to purulent and back to mucoid with progressive alterations in the underlying periosteum and bone and polyp formation in the mucosa.

In considering the extension of nasal infection Lederer[5] emphasizes the fact that periostitis and osteitis are preliminary to orbital, intracranial, oral, and some venous extensions. Inasmuch as the sinus mucosa is intimately attached to the underlying periosteum, an allergic process in the submucosa may similarly sensitize the periosteum and bone. The latter structures are unavoidably affected by the sinus contents particularly when the acute edema of the hyperplastic lining builds up sufficient intrasinus pressure to extrude polypoid membrane through a small sinus ostium into the middle meatus. In the same manner that the skin of the body has been considered the shock organ for an allergic process in other epithelial tissues in the body, so the sensitized periosteum and bone of the sinus may evoke a "shock organ" response in other bones of the body. This intranasal pressure resulting from the edema is known at times to produce a profound reflex effect upon the peripheral and central nervous systems. The pressure and toxic effect is exerted upon branches of two cranial nerves, the olfactory and trigeminal, and sympathetic fibers chiefly from the superior cervical sympathetic

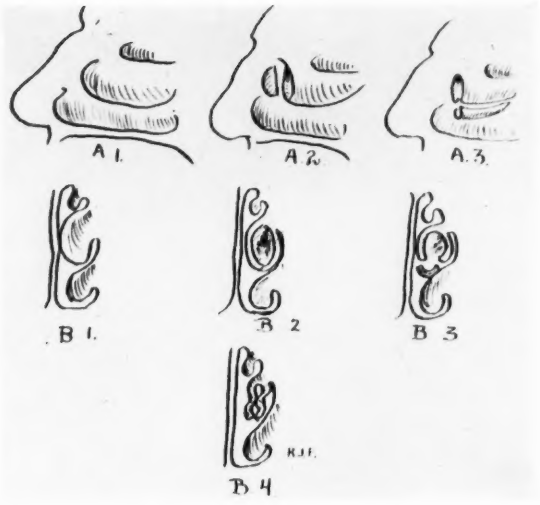


Fig. 2.—Diagrammatic representation of the steps in the plastic reduction in size of a bulbous middle turbinate. The anterior tip is removed, and the lower edge is cut away so that when the walls are pressed together, it will not hang low enough to fall against the inferior turbinate.

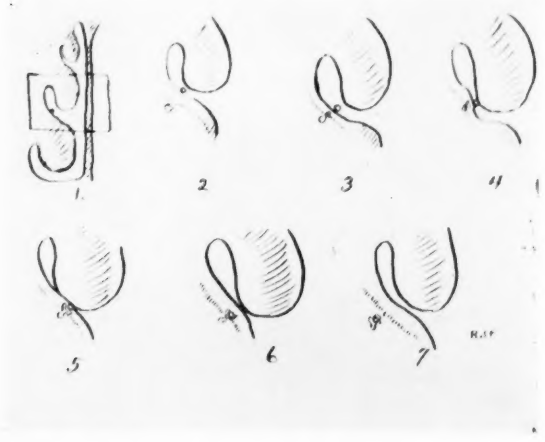


Fig. 3.—Diagrammatic representation of foreign protein penetration of nasal mucosa under influence of contact and pressure of opposing surfaces. Passing through the basement membrane a leucocyte reaches the foreign substance and is carried to the reticuloendothelial system of the tunica propria.

plexus. Is it not conceivable that, regardless of reflexes, when these nerves are compressed in an epithelium reacting to undigested foreign protein, they themselves may develop a neural hypersensitivity? If so, other nerves in the body would become shock organs to this reaction and could undergo localized sensory and motor neuritis or slowly degenerative diseases of the nervous system characterized by edema and vacuolization. Neurological cases are being more carefully studied from the nasal and allergic standpoints.

So well appreciated is the association of bronchitis, bronchiectasis, and bronchial asthma to chronic hyperplastic ethmoiditis that this extension will only be indicated. Roentgenologists are suggesting sinus investigation in asthmatics with roentgenological evidence of pulmonary involvement. The inhalation of dusts directly into the lung as the result of nasal obstructions and the nasal-pulmonary reflexes of the sympathetic nervous system indicate some of the direct connections between nasal disease and pulmonary allergy.

Nasal lymphatics have been studied by Larsell and Fenton.[6] There are four to six large lymphatic collectors in the middle meatus which join with lymphatics of the upper surface of the inferior turbinates and the lower surface of the middle turbinates.

There is extension to the deep superior cervical, to the bronchial and mediastinal lymph nodes. The combined path of lymph nodes joins with those of trachea and lymphatic duct, which reach the right side of the heart and the pulmonary bed.

Nasal extension to the gastrointestinal tract is a broad subject. Besides lymphatic and hematogenous extension there is the day and night swallowing of postnasal secretion. This secretion may fall upon the gastric mucosa when the secretion of hydrochloric acid is at its lowest concentration. It may fall upon gastric mucosa overdistended by large meals, from which are being absorbed undigested protein fractions. Allergens in the nose often produce gastric symptoms and allergenic food may suddenly or gradually produce allergic changes in the nose.

Following the hay fever season, many allergic individuals should have a bilateral antrum irrigation to eliminate the pollen and mucus incident to the four weeks of sneezing. Harris and Thomas[7] state: "X-ray examination may reveal cloudy antra or cloudy frontal sinuses, but the presence of pus can be determined only by irrigation." Proetz[8] has stated: "On account of the nature of mucus,

sinus secretion may have so great viscosity and high surface tension as to remain adherent to the sinus wall and not have a fluid level on the floor of the sinus."

A few days ago a 14-year-old girl showed both antra to be filled with such secretion, although it had been unsuspected during antiallergic treatment for extreme eczema of the hands for the past five years. In purely allergic processes there may be little or no x-ray or transillumination evidence of dust or secretion in the sinus, but the determination of purulent secretion can be made by irrigation. Its removal relieves the patient of the biochemical build-up preparatory to the allergy of the following season.

Since employing prolonged postnasal irrigations with normal saline solution at 100°, it has been surprising to find the frequent association of nasal infection with allergic states. Small, firm, yellowish plugs from blocked ethmoidal sinuses are obtained after two to four quarts of irrigating fluid have loosened the material. Such irrigations are frequently followed by marked relief from subjective symptoms and in acute allergic phenomena there may often be an extensive emptying of purulent secretion which has been retained beneath the shoulders of the turbinates. The technic of these irrigations is as follows : The nose and uvular regions are sprayed with a solution of 1 per cent pontocaine in 3 per cent ephedrine hydrochloride solution. A lozenge containing about ¼ gr. anesthesin is dissolved on the back of the tongue. The postnasal irrigating tip, provided with three tiny openings, is attached by rubber tubing to a three-way connector. One of the remaining openings is used for a thumb control of the stream. The other is attached by rubber tubing to an irrigating can containing two quarts of normal physiological sodium chloride at a temperature of 108° F. Using reflected light, one passes the postnasal tip beyond the depressed tongue into the postnasal space. The patient's head is brought forward together with the control hand on the irrigating tip. The patient breathes through his mouth, the tubing is opened, and the solution is permitted to run into a lap basin through each nostril successively. The removal of the finger from the three-way connector gives immediate control of the stream in case the patient wishes a breathing spell. The average patient is very cooperative inasmuch as no negative or positive pressure alterations are produced either in the nose, sinuses, or eustachian tubes. There have been no cases of extension to the ear such as occur occasionally following other forms of irrigations, particularly where suction is used. The only irrigations which have been comparably effective, as compared to this procedure, have been the use of anterior nasal irrigations with

warm physiological sodium chloride solution directed against the anterior tip of the middle turbinate. After both anterior and postnasal irrigations, the nose is dried as completely as possible by suction through a small cannula applied directly to the mucus.

The postnasal irrigation directs the stream of water under the overhanging shoulders of the turbinates and reduces the surface tension of dried secretions lying over the ostia of the sinuses. Much of this material is probably forced into the nose with considerable pressure during the excessive and violent blowing of the nose, which often seems to be a habit of the hypersensitive individual. If correction of the allergic state is accomplished by any means whatsoever, positive pressure nose blowing will contribute to its recurrence. For the allergic individual there is no safe method of nose blowing.

A girl 12 years of age operated upon three years ago by submucous resection and plastic reduction in the size of both middle turbinates has refrained from nose blowing since that time, and her nose has a normal appearance now, at the age of 15 years, although during the three years prior to operation she suffered numerous head colds in the winter, nasal obstruction daily, and severe hay fever with asthmatic breathing during the pollen seasons. Her return to normal is largely attributed to her fine cooperation in refraining from blowing the nose.

The preventive treatment of general allergy extends widely into the field of general medicine. The place of diet, vitamin and endocrine therapy, the use of autogenous and stock vaccines, and other phases of the allergic regimen need no consideration in this paper.

The treatment of sinus infections in infancy and childhood has been given more consideration since Proetz has advocated the use of the displacement method by the instillation of weak vasoconstricting solutions. The infected sinuses should not be allowed to lapse into a state of chronic infection following acute respiratory diseases and especially after pertussis and exanthematous diseases. All pollens and irritants should be cleaned out from the nose after seizures of allergic reaction. The exclusion of the offending inhalants is part of the allergic regimen.

Whereas prolonged infection usually leads to irreversible changes in the nasal and antral mucosa, allergic changes more often return toward the normal condition when the hypersensitivity has been relieved. Therefore, "allergy surgery" is not destructive in type, but is eliminative and restorative. It can be said to include resection of the nasal septum; removal of septul spurs; plastic reduction of

the size of the middle turbinates, or removal of their anterior tips, or repositioning of the middle turbinates to free contact points; intranasal window operation; partial removal of a hypertrophied posterior tip of the inferior turbinate; adenoidectomy; and intranasal exenteration of the ethmoidal labyrinth, which may be well accomplished by the Mosher technic. Mulberry hypertrophy of the posterior tip of the inferior turbinate may require reduction in its size and some surgeons use submucous exposure of the inferior turbinal bone and shave off a portion of its mesial surface to increase very slightly its clearance from the septum without producing overventilation.

In general, nasal surgery beyond the above limitations cannot be considered as "allergy surgery." Hill[8] aptly states: "Excluding cases of pollinosis, surgery is justified despite the presence of allergy only if the clinical picture after careful study would suggest operative interference anyway if the case were not allergic. In other words, we are probably dealing with sinuses in which bacterial involvement has occurred secondarily to the allergic condition. Generally, in these cases, a combination of allergic treatment and rational surgery gives the best results."

In any form of allergic manifestation in which the patient is a good operative risk, chronically thickened antral lining and polyps should be removed by the Caldwell-Luc operation if inspection of the nasal secretion or antral irrigation shows pus to be present, as the pathologic sections will usually show localized abscesses present in such antral linings.

CONCLUSIONS

1. By its anatomy and function, the nose, especially the ethmoidal labyrinth, is an ideal organ for holding the former inciting causes of hypersensitivity and for reacting to the new assaults of allergic excitants, whether blood-borne or air-borne.

2. The mechanical causes which interfere with the development of physiologic negative pressure in the nose and accessory sinuses during inhalation delay normal sinus emptying and the movement of the sheet of mucus.

3. In general, those bacterial and nonbacterial substances which have the best opportunity in the nose to incite the allergic reaction are those which either become impinged by pressure between opposing mucosal surfaces of turbinates and other nasal walls, or which are locked under pressure in small sinus cavities. The customary prac-

tices of nose blowing are harmful in proportion to their frequency, the degree of intranasal positive pressure produced, and the amount of foreign substance forced under the turbinates and into the sinuses at the time of blowing.

4. "Allergy surgery" is nasal surgery aimed at the correction of intranasal deformities so as to simulate the normal arrangement; allowance for sufficient clearance for the turbinates, so that changes in temperature and humidity will not interfere with normal nasal function; adequate sinus ventilation and drainage where secretion has formerly accumulated; removal of polypoid membrane, when prolonged reaction has made its condition irreversible to normal; exenteration of ethmoid labyrinth in certain cases showing the more serious forms of allergy, such as bronchial asthma, allergic arthritis, etc., if there is evidence of chronic nasal disease.

5. All nasal and body infections should be eliminated with the same or greater care than in normal individuals. Similarly, persons with nasal or other infections should be carefully studied from an allergic point of view even though the evidences may not be obvious.

15701 Detroit Ave.

BIBLIOGRAPHY

1. Richou, R.: Rev. immunol. 3:25 (Jan.), 1937.

2. Kuhn, H. A., and Linton, L. D.: Allergy in Relation to Otolaryngology, Annals of Otology, Rhinology and Laryngology 51:164 (March), 1942.

3. Proetz: Applied Physiology of the Nose, St. Louis, 1941, Annals Publishing Co.

4. Hansel: Allergy of the Nose and Paranasal Sinuses, St. Louis, The C. V. Mosby Co., 1936.

5. Lederer, Francis L.: Diseases of the Ear, Nose and Throat, Philadelphia, 1939, F. A. Davis Co., p. 416.

6. Larsell, Olof, and Fenton, Ralph A.: Lymphatic Pathways From the Nose, Arch. Otolaryng. 24:696-713 (Dec.), 1936.

7. Harris and Thomas: Allergy in Clinical Practise, Philadelphia, 1941, J. B. Lippincott Co.

8. Hill, Frederick: Indications for Surgery in Sinusitis, Tr. Am. Acad. Ophth., Oto. & Laryng., p. 158, 1938.

LVIII

ACUTE FULMINATING OSTEOMYELITIS OF THE FRONTAL BONE: A REVIEW AND A PREDICTION*†

GUSTAVE B. FRED, M.D.

BOSTON

This article is a review of 40 cases of acute fulminating osteomyelitis of the frontal bone occurring on the wards of the Massachusetts Eye and Ear Infirmary during the past 10 years (1931-1941). It does not include chronic cases, traumatic cases, cases which on admission presented a draining fistula, or sinusitis cases which subsequently developed osteomyelitis after sinus surgery.

Acute fulminating osteomyelitis of the frontal bone is a virulent infection which complicates acute frontal sinusitis and spreads rapidly and wildly to the adjacent bone. It frequently follows swimming and diving, and is marked by profound toxicity. Its earliest and most characteristic sign is pitting doughy edema of the forehead, often up to the hairline. The disease is very often fatal, usually terminating in brain abscess or meningitis. The etiological organism is usually Staphylococcus aureus, sometimes Streptococcus haemolyticus.

There are two types of acute osteomyelitis of the frontal bone: (1) the fulminating type and (2) the localizing type. The latter is less virulent, tends to burn itself out, becomes localized and forms sequestra. This form of osteomyelitis can be dealt with very much less radically at operation than the fulminating type. Some cases of the fulminating type will perhaps develop into the localizing type if the surgeon elects to use delaying tactics, such as incision and drainage, the ethmoid or Lynch frontal operation. Those that do not develop into the localizing type often go on to meningitis or brain abscess unless the frontal bone is removed.

When one sees a patient for the first time, there is unfortunately no rule or criteria to follow in determining at that examination

*From the Department of Otolaryngology of the Massachusetts Eye and Ear Infirmary.

†Presented before the American College of Surgeons at Boston, Nov. 7, 1941.

whether that particular case of osteomyelitis will eventually develop into the fulminating type or the localizing type. If one is lucky enough to pick successfully the localizing type of case, and waits for sequestration, the patient is indeed fortunate. If, however, the doctor guesses wrong, the case usually ends in a tragedy. Upon that point I wish to lay particular stress because, seen in that light, all cases of acute osteomyelitis of the frontal bone in this series were considered and dealt with from the beginning as if all were the fast-spreading, destructive fulminating type. Early radical removal of the frontal bone was therefore the procedure followed at the Infirmary.

An exception to this rule was made in those cases where trauma was the etiological factor. These cases are less virulent and should be allowed, under observation, to sequestrate. They usually do well after the sequestrum is removed.

One of the cardinal principals recognized in the diagnosis of the disease is that pitting doughy edema of the forehead is the first and most reliable sign of osteomyelitis of the frontal bone. It precedes the X-ray by at least a week or ten days and is due to the thickening or separation of the periosteum from the frontal bone. It is indicative of early microscopic osteomyelitis of the bone. This edema, in the words of Mosher, is a rough guide to the extent of the osteomyelitis, and demonstrates approximately how much bone should be removed. This sign alone has been accepted by us as sufficient evidence for the diagnosis of acute fulminating osteomyelitis of the frontal bone.

It has been the practice at the Infirmary not to wait for a positive X-ray report of osteomyelitis when such edema is present, because we have regarded this sign as pathognomonic of the disease. A positive X-ray means gross destruction of bone. This takes time and gives the disease an opportunity to spread and cause further complications.

Mackenzie[1] in his original article recommended a wide radical removal of the diseased osteomyelitic bone. This principle was followed at the Infirmary in that from one to one and one-half inches of normal appearing bone were removed together with the grossly diseased bone. This was accomplished through an inverted T-incision, with the base of the T just above the eyebrows. The vertical portion of the incision reaches usually to the hairline or as far as is necessary to reach the pathology. Burr holes are made with an electric drill in normal bone at least one inch outside the osteo-

myelitic bone. The burr holes are joined together by Gigli saw or Devilbiss rongeur, and the entire bone removed if possible in one piece. The bone removed usually extends beyond the lateral limits of each frontal sinus and preferably to each external angle of the frontal bone. The anterior and posterior walls of each frontal sinus should be removed. The floor of the frontal sinus is removed if diseased, or if the ethmoid exenteration is contemplated; otherwise it is left undisturbed. An attempt should be made to save the orbital rim if it is not diseased. It has been our practice to remove both sides of the frontal bone in the acute fulminating type even though only one side is diseased. This plan was particularly followed when edema and X-ray revealed osteomyelitis near the midline. Formerly at the Infirmary only the one definitely osteomyelitic side was removed. A second and a third operation was often required to chase the spreading disease across the midline. These cases often advanced beyond control and were lost. Experience has taught us that the best chance of cure is by wide removal of the entire frontal bone at the first operation. All bone which is separated from its periosteum during the operation is removed with rongeurs. This prevents sequestra from forming at the edge of the bony defect. The incisions were left wide open and allowed to epidermize over. After a period of no less than three months, plastic closures with bone transplants were performed for cosmetic purposes.

The above ideas and plan of attack were inaugurated and followed at the Infirmary at the suggestion and under the guidance of Mosher,[6, 7, 8] to whom full credit is hereby given.

There were 40 cases of acute fulminating osteomyelitis of the frontal bone admitted to the Massachusetts Eye and Ear Infirmary during the ten-year period between 1931 and 1941, an average of four cases a year. They were divided into two five-year groups. During 1931 to 1936 there were 12 patients of whom six died, a mortality of 50 per cent. In 1936 to 1941 there were 28 patients with six deaths, a mortality of 21.4 per cent. The total mortality for the 40 patients was 30 per cent.

The mortality of 50 per cent in the first five-year group was due, I feel, to delayed and inadequate surgery. In those days, our ideas had not crystalized to the point of removing the entire frontal bone. An ethmoid exenteration or an obliterative frontal sinus operation was usually performed with piecemeal removal of the osteomyelitic bone as it appeared. In that way the osteomyelitis spread and required multiple operations. The operations were also not done early enough in the disease in that group.

The 21.4 per cent mortality in the latter five-year period is a marked improvement due to early radical removal of the entire frontal bone in most of the cases. The 30 per cent mortality of the entire series compares very favorably with larger percentages obtained by other clinics. Gerber in 1909 had a 100 per cent mortality; Mackenzie in 1915, a 83 per cent mortality; Bulson in 1926, a 68 per cent mortality; Wilensky in 1931, a 50 per cent mortality.

There were 27 males and 13 females in the list, a ratio of 2:1. The youngest was 4 years of age, the oldest, 48 years. Osteomyelitis is a disease of the young. One-half of the cases in this series occurred during the second decade of life (tenth to twentieth year); 11 (27.5 per cent) cases occurred during the third decade. There were 6 cases in the first decade; none in the fourth decade; and 3 in the fifth decade. Three-fourths of all the cases, therefore, took place between the ages of 10 and 30 years.

Young individuals also indulge more frequently in swimming and diving, a prolific cause of osteomyelitis, especially when associated with acute coryza. This was found in our series. Almost one-half of the cases (17 or 42.5 per cent) occurred during July (7), August (6) and September (4) which coincides with the swimming season. The remainder were distributed as follows: January, 3; February, 2; March, 4; April, 3; May, 1; June, 1; October, 3; November, 3; December, 3.

Osteomyelitis was found exclusively at operation on the left side in 8 cases; on the right side in 4 cases. Twenty-eight cases (70 per cent) at operation revealed osteomyelitis on both sides, with a mortality of only 6 (21.4 per cent). The large incidence of osteomyelitis upholds the contention that it is best, especially when the osteomyelitis approaches the midline, to remove both sides of the frontal bone as a routine. In the latter years of the series, the entire osteomyelitic bone with an edge of surrounding normal bone was removed in one piece. When this was done, the mortality in the series decreased considerably.

The best chance for cure is by removal of the entire frontal bone at the first operation. This plan was attempted in the cases that fell into the last five-year group. This was not done in the first five-year group with the result that many two- or three-stage operations had to be performed for the removal of advancing osteomyelitis. The mortality of only 13.6 per cent (3 deaths out of 22 patients) when the entire frontal bone was removed confirms this fact when compared with the greater mortality of 57.1 per cent (4 deaths out

of 7) and 45.4 per cent (5 deaths out of 11) where less radical procedures were used.

Formerly it was taught that the ethmoid sinuses must be exenterated when osteomyelitis of the frontal bone was encountered. However, owing to the length of the operation the ethmoids were not removed in many of the cases. To our surprise most of the cases recovered without further operative interference. The ethmoids were not mentioned in the records in 9 cases (6 recovered). I have presumed that when the ethmoids were not mentioned they were not exenterated. Therefore, 24 cases had no ethmoid exenteration with recovery of 18. In 16 cases with exenteration, 10 recovered. One can conclude, therefore, that most cases will recover without ethmoid exenteration.

In 5 cases the dura was torn at operation when adherent osteomyelitic bone was removed. In one of the cases cerebrospinal fluid escaped. All 5 cases recovered. While this accident is unfortunate and appears formidable, it is not necessarily followed by meningitis.

Extradural abscess alone (12) or associated with subdural abscess (1), with brain abscess (7), or with both (1) was found in 21 cases (52.5 per cent). Brain abscess alone without extradural abscess was found in 2 cases and subdural abscess alone without extradural abscess in 1. Extradural abscess, brain abscess, and subdural abscess were found in 24 cases, or 60 per cent of the total. These large percentages explain why osteomyelitis of the frontal bone ends fatally in so many cases and why it is necessary to deal with them so radically at operation. In only 16 cases was there no extradural, subdural, or brain abscess found; in other words the dura was normal in only 16 cases out of the 40. One can say therefore that dural pathology should be expected and is found in the majority of cases of acute fulminating osteomyelitis of the frontal bone.

Pathology inside the dura (subdural and brain abscess) was found in 12 cases, or 30 per cent of the total. Ten frontal lobe brain abscesses (25 per cent) were found among the 40 cases. Four of the ten recovered (40 per cent).

One-half of the 40 cases on admission revealed a negative X-ray report for osteomyelitis. The report in these cases usually read pansinusitis. The frontal bone was positive in 14 cases; the anterior wall of the frontal sinus positive in 4; the X-ray was lost in 2 cases; and the X-rays were not positive for osteomyelitis in 20 cases. Eleven of these on repeated X-ray finally developed osteomyelitis of either the frontal bone or the front face of one of the frontal sinuses.

Nine never became positive for osteomyelitis at any time. This was due to early removal of the osteomyelitic bone when pitting edema of the forehead was present. All such cases by pathologic report after removal revealed osteomyelitis. This also bears out the contention that edema of the forehead is the first and the most reliable sign of osteomyelitis. It preceded the X-ray in 12 cases of this series by an average of twelve days. The histories in the other cases were not clear on this point and were therefore not included. The shortest period was four days, the longest twenty-two days.

In admission and interval X-rays the frontal bone was positive in 19 cases.

All of the 10 cases of anterior wall osteomyelitis recovered, but it should be noted that one had extradural abscess, and another brain abscess. Therefore, evidence of osteomyelitis confined to the front face of the frontal sinus must be considered potentially dangerous.

Nine cases never became positive for osteomyelitis by X-ray because of early operation. All presented doughy pitting edema of the forehead. There was a mortality of 4 (3 brain abscesses and 1 subdural abscess). This high mortality (44 per cent) should be looked upon as evidence against delaying operation when edema is present, even though X-ray evidence of osteomyelitis is lacking. X-rays were lost in 2 cases.

Chemotherapy (sulfapyridine and sulfanilamide) was used in 15 cases with 10 recoveries in the last four years of the series. The drugs played only a minor part in the recovery of the cases. Sulfathiazole and sulfadiazine, the two effective chemotherapeutic agents against Staphylococcus aureus, were not available at the time. What their effect on this disease will be only the future can tell.

I hope it will not be considered presumptuous if I hazard a prediction as to the future treatment of acute fulminating osteomyelitis of the frontal bone. I realize it is risky to predict the future, particularly when one does so with a very limited experience. I therefore approach this task with some misgiving. The period before 1941 might be appropriately designated as 1941 *B. C.* (before chemotherapy), whereas, the period after 1941 could be christened *A. D.* (after diazine). I am convinced that chemotherapy will surely modify, even revolutionize, the treatment of osteomyelitis.

The greatest use of chemotherapy will no doubt be in the prevention of the disease, provided two conditions are satisfied. First, the drug sulfadiazine must be used early in the disease and in

adequate dosage. The blood level should be kept at no less than 20 mg. per cent (it is conceivable that a lower concentration might suffice) for optimum results during treatment in the early stages. Second, incision and drainage through the floor of the frontal sinus must be instituted very early in the disease along with chemotherapy. An incision similar to the one usually made for an external ethmoid exenteration, but much smaller, should be made, and the periosteum separated only enough to allow removal of a small part (1 cm.) of the floor of the frontal sinus for drainage of pus. Extreme care should be exercised not to traumatize, separate, or suck out the mucous membrane lining the frontal sinus. No ethmoid cells should be removed even though the X-ray shows them involved. The best plan is to do as little as possible in an operative way consistent with free drainage from the frontal sinus. The maxillary sinus on the same side should likewise not be irrigated or operated upon at this initial stage of the disease. If these two conditions are satisfied I feel that most cases of potential osteomyelitis of the frontal bone will be prevented. Chemotherapy alone (i.e., without incision and drainage) is, in my opinion, not enough in the average case, though no doubt some patients will recover on chemotherapy alone.

CASE 1.—A 13-year-old girl was recently admitted to the Massachusetts Eye and Ear Infirmary with a history of severe coryza of ten days' duration. Both eyes were edematous, the left closed tight with marked limitation of motion. She was toxic (temperature, 103°) and presented doughy pitting edema up to the hairline which had been present for six days previous to admission. The case therefore was a potential case of acute osteomyelitis of the frontal bone. The X-ray report was pansinusitis on the left. Sulfadiazine therapy together with incision and drainage through the floor of the left frontal sinus were immediately instituted. No orbital abscess was found. The eye movements became normal in three days, and the edema over the forehead disappeared completely in six days. She was discharged cured from the hospital in sixteen days. I feel very certain that in the days before chemotherapy this case would have been treated by removal of the frontal bone.

If a case is first seen in a more advanced stage of the disease, it may already present X-ray evidence of osteomyelitis of the front wall of the frontal sinus. Notwithstanding this, I would still rely on incision and drainage plus chemotherapy, provided there is no evidence of meningitis, brain abscess, extradural abscess or sepsis. Of course, it will be necessary to have repeated X-rays, eyeground examination, spinal puncture and neurological examination for the question of dural pathology. To be sure, one will have to be on the lookout for chills, septicemia, pulmonary infarcts and high white counts for evidence of sepsis.

If these signs are present it will be necessary to remove part or all of the frontal bone and deal with the complication. If they are

absent, I would continue with prophylactic measures as enumerated above, in the hope and expectation that sequestration and possibly recalcification would result.

CASE 2.—A very toxic patient, 25 years of age, was admitted to the Infirmary complaining of a very severe headache, temperature of 103°, with both eyes edematous and closed, and X-rays revealing bilateral pansinusitis. This followed an acute coryza of one month's duration. Sulfathiazole and sulfadiazine were not available at the time. Sulfapyridine plus incision and drainage through the floor of each frontal sinus were immediately instituted. Despite these measures osteomyelitis of the anterior wall of the right frontal sinus was reported by X-ray. He was advised to have the frontal bone removed but refused, and was discharged from the hospital against advice. On three separate admissions the same advice was given and three times refused. Treatment outside the hospital consisted of shrinkage of nasal mucous membrane, removal of the anterior tip of the left middle turbinate, and bilateral antrum irrigations with the result that the progress of the disease was arrested and the osteomyelitic area became recalcified as shown by X-ray. This demonstrates clearly that osteomyelitis can heal by calcification.

If a case is seen very late in the course of the disease for the first time it will probably reveal definite osteomyelitis of the frontal bone above the upper limit of the frontal sinus. The treatment here would depend on the condition of the patient. If I had a very sick patient with signs and symptoms of a complication, I would certainly remove sufficient frontal bone to deal properly with the complication. If signs of complications are absent there would then be no great emergency and no need for haste. I would therefore use chemotherapy plus incision and drainage through the floor of the frontal sinus and over any scalp abscesses present with the hope that the progress of the disease could be arrested and sequestra would form. These can be subsequently removed. There is, of course, a limit to the time chemotherapy should be used. There is the danger of some operators being lulled into a false sense of security with this drug and never operating. Sound judgment and long experience will be required in the future to evaluate the drug properly.

The question of primary or secondary closures comes up in those cases where it is nececssary to remove the frontal bone. At the Infirmary we have always used the inverted T-incision and have packed it wide open, having in mind that intracranial complications are always just around the corner. However, we are now in a favorable position with the use of chemotherapy where I feel that the coronal incision and primary closure with adequate drainage can be used. Recently a case following a history of swimming was admitted in a comatose condition to the Massachusetts General Hospital with osteomyelitis of the frontal bone up to the coronal suture, hemiplegia, and bilateral blindness from increased intracranial pres-

sure of long duration. A diagnosis of osteomyelitis of the frontal bone and frontal lobe brain abscess was made. Sulfadiazine was prescribed for two days, after which the osteomyelitis bone was removed through a coronal incision inside the hairline. The brain abscess was needled, 6 gm. of sulfanilamide powder dusted over the dura, and the incision closed with drainage at the two extremities. Sulfadiazine was given by mouth. The incision healed by first intention. Ten days later a cone of brain cortex together with the upper half of the abscess capsule was removed through a separate skin incision over the abscess. A week later the bottom half of the abscess capsule was dissected from the brain and 6 gm. of sulfanilamide again placed in the cavity. At the last report the patient was recovering with some return of sight. This case demonstrates that primary closure of the incision is feasible.

SUMMARY AND CONCLUSIONS

1. Forty cases of acute fulminating osteomyelitis of the frontal bone occurring on the wards of the Massachusetts Eye and Ear Infirmary from 1931 to 1941 are reviewed.

2. The low mortality of 30 per cent was due to early removal of the frontal bone in most of the cases.

3. There were two males to one female.

4. Almost one-half of the cases occurred during the swimming season.

5. Edema preceded the X-ray by an average of twelve days in 12 cases.

6. Osteomyelitis was found at operation on both sides in 70 per cent of the cases.

7. The dura was torn in five cases with recovery.

8. Most cases recovered without ethmoid exenteration.

9. Extradural abscess, subdural abscess and brain abscess were found in 60 per cent of the cases.

10. On admission one-half of the cases revealed no osteomyelitis by X-ray.

11. Almost one-fourth of the cases were never positive for osteomyelitis by X-ray, due to early removal of the frontal bone.

12. All cases of osteomyelitis of the anterior wall of the frontal sinus recovered.

13. The coronal incision with primary closure will probably be the method of choice in the future.

14. It is my opinion that most cases of potential osteomyelitis of the frontal bone will be prevented in the future if adequate chemotherapy plus incision and drainage through the floor of the frontal sinus are instituted early enough in the disease.

15. It is my belief that the operation for removal of the frontal bone will be used in the future only on those rare occasions where proper prophylactic measures have not previously been used—which is another way of saying that the operation, as a method of choice, is in my opinion, on the way out.

520 BEACON ST.

I wish to thank Dr. H. P. Mosher and Dr. Leroy A. Schall for their helpful suggestions and cooperation in the preparation of this paper.

BIBLIOGRAPHY

1. Mackenzie, D.: Osteomyelitis of Frontal Bone, J. Laryng. & Otol. 28:6, 1913-14; 42:293, 1927.

2. Bulson, A. E., Jr.: Further Observations on Spreading Osteomyelitis of the Skull, Tr. Am. Acad. Ophthal. & Otol. p. 102, 1925.

3. Skillern, R. H.: An Extensive Case of Acute Osteomyelitis of the Frontal and Superior Maxillary Bones Complicating Sinusitis, Operation, Apparent Recovery, Sudden Death, ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY 38:1022 (Dec.), 1929.

4. Furstenberg, A. C.: Osteomyelitis of the Skull: The Osteogenetic Processes in the Repair of Cranial Defects, ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY 40:996 (Dec.), 1931.

5. Wilensky, A. O.: Association of Osteomyelitis of the Skull and Nasal Accessory Sinus Disease, Arch. Otol. 15:805 (June), 1932.

6. Mosher, H. P., and Judd, D. D.: An Analysis of Seven Cases of Osteomyelitis of the Frontal Bone Complicating Frontal Sinusitis, Laryngoscope 43:153, (March), 1933.

7. Mosher, H. P.: Osteomyelitis of Frontal Bone, J. A. M. A. 17:942 (Sept. 19), 1936.

8. Mosher, H. P.: Osteomyelitis of Skull, Surg., Gynec. & Obst. 69:417 (Nov.), 1939.

LIX

SOME CAUSES FOR FAILURE IN FRONTAL SINUS SURGERY*

ROBERT LINCOLN GOODALE, M.D.

BOSTON

The surgical management of frontal sinus infection probably calls up as much discussion as any other nose and throat condition. This is probably due to the varying experiences of the different operators.

At the Massachusetts Eye and Ear Infirmary, there has been a growing dissatisfaction with the results of the procedures which we have followed. In order to find out why the results have not been as satisfactory as we had expected, this present review of cases has been undertaken. I would like to preface the study with the remark that at the Eye and Ear Infirmary frontal sinus surgery has been undertaken only in such cases as urgently required it; such as an empyema of the sinus which would not drain by more conservative procedures, orbital complications, extension of the infection into the frontal bone, or intracranial complications. It also is a principle of procedure that the choice of operation should be left to the judgment of the member of the staff who is responsible for the case. It is felt that no standardization of procedure is justified. Each case presents its own individual problem. In this paper, I assume that the surgeon responsible had evaluated the situation so that, under the circumstances, the operation was the type best suited to the case. The operations devised for the frontal sinus have all been described in the literature, and it is not the purpose of this article to review those procedures.

The word "failure" suggests that we expect a frontal sinus operation to remain 100 per cent successful for the rest of the patient's life, but I do not think that this is a fair expectation. After all, the patient presents us with a problem in which his life is already in danger or, at best, he is exposed to infection which is causing more and more disability. The prime object of the operation may

---

*From the Department of Otolaryngology of the Massachusetts Eye and Ear Infirmary.

be to save the patient's life and, if that is done, the operation is successful. If his health is improved and he returns to his normal life, even for a limited period of time, we cannot say that the operation has been a failure. We expect a great deal from our operations. We would like to have the patient go on through life with no further trouble in that sinus. When the frontal sinus becomes again the site of infection, requiring reoperation, it is natural to feel that the first operation has failed.

This paper is a short review of frontal sinus operations which were done at the Massachusetts Eye and Ear Infirmary in the eight years from 1933 to 1942. It is not an attempt to criticize the type of operation which was undertaken in the beginning, nor to draw conclusions as to the relative value of these operations. It is rather to discover, if possible, what anatomical or pathological conditions were found at the second operation which could account for the necessity for reoperating.

During the eight years which I have reviewed, there was a total of 190 cases in which external operations on the frontal sinuses were performed. Due to some difficulty in finding records, eight cases had to be eliminated, bringing the number which I am reporting down to 182. In these cases the diagnoses on admission were distributed as follows: infection, 123; mucocele, 18; acute osteomyelitis, 33; osteoma, 2; meningioma, 1; cholesteatoma, 1; unclassified cyst, 1; fracture, 1; primary carcinoma, 2.

The cases of osteomyelitis are being treated in another article by Dr. G. B. Fred and do not form a part of this study. I am also leaving out of consideration the cases of carcinoma, osteoma, meningioma, fracture and unclassified cyst, so that the only cases for consideration are those in which there was infection primarily, or a mucocele.

In the first group, in which infection was the diagnosis, there were 38 recurrences requiring operation, or 30.8 per cent. In the cases in which there was a mucocele, there were four recurrences out of 18 cases, or 22.2 per cent.

I would like to take up these two groups separately. In the first group, in which infection alone was the consideration, it was found at reoperation that certain pathological or anatomical considerations recurred with considerable frequency. It is interesting to note that one operator will explain his failures on one of these conditions, whereas another may find another factor as his difficulty. Adhesions always seem to be a problem, but a problem which can

be frequently taken care of in the postoperative treatment. They are, I think, the first cause for failure which most of the operators at the Eye and Ear Infirmary think of when questioned. Undoubtedly, they are potentially the greatest cause for failure. In reviewing the findings at reoperation, the following conditions were found to be present.

In the group of infections there were three cases of mucocele, three of pyocele, four of sequestrum, five of osteomyelitis, one of brain abscess, 15 of frontoethmoid cell or orbital extension of ethmoid, 16 of adhesions, and 17 of remnants of frontal sinus floor.

In the group of mucoceles there were four cases of recurrence of mucocele, one case each of adhesions, ethmoid remnants, and remnants of the frontal floor.

It will be seen that adhesions, remnants of the frontal sinus floor, frontal ethmoidal cells, and ethmoid extensions into the roof of the orbit are found in the greatest number of cases. In many of the cases, more than one condition was present. It is interesting to note the types of operations that were performed. At the Massachusetts Eye and Ear Infirmary, the term "Lynch operation" is commonly used to describe that operation in which the floor of the frontal sinus is removed, and in which a complete ethmoidectomy and drainage of the sphenoid is also included. The term "obliterative" should be explained. It entails the removal of the floor of the frontal sinus and ethmoidectomy, and a removal of the anterior wall of the frontal sinus. It necessitates a complete removal of all the mucous lining of the sinus. It includes removal of the posterior wall if that is diseased. In the opinion of Dr. Mosher and some other members of the staff, the posterior wall should be removed routinely.

The result which is desired in this operation is a complete obliteration of the frontal sinus cavity, and it is considered a certain cause for failure if any pockets of infection are left behind, or any remnants of mucous membrane remain to form mucoceles. Four of the patients had a Killian type of operation. Only one of these has had no recurrence. Two developed pyocele and required an obliterative second operation. The fourth case was revised, and in this the three most common causes for failure were found: scar tissue, ethmoid cells, and remnants of the frontal sinus floor.

In the 123 patients admitted with the diagnosis of infection, four had the Killian type operation, 106 the Lynch type, and 13 the

obliterative. There were three recurrences in those who had the Killian type operation, 30 in those who had the Lynch type, and five in those who had the obliterative. Two of the three cases of recurrence after the Killian operation had reoperation by the same technic and one by the obliterative type of operation. Of the 30 recurrences following the Lynch operation, 23 had reoperation by the same technic and 7 by the obliterative. The five cases of recurrences after the obliterative type of operation had reoperation by the same technic.

In the 18 patients who were admitted with a diagnosis of mucocele, 11 had the Lynch type of operation and seven the obliterative. All the recurrences were in those who had had the Lynch operation. Three of these were successfully treated by revising the original operation; in other words, removal of scar tissue, remnants of the floor and reopening the frontal sinus into the nose was sufficient.

It will be seen from the statistics that in many cases the procedure at reoperation was to remove certain anatomical structures; namely, remnants of the frontal sinus floor and ethmoidal cells. The frequency with which these were found at reoperation emphasizes the need for fully understanding the important points involved in an external operation on the frontal sinus. Unless all pockets in the frontoethmoid region are eliminated by removal of bony partitions and small shelves of bone which form ledges in the floor of the sinus, reinfection will recur sooner or later in these areas. Scar tissue will have a greater opportunity to form barriers within the sinus and conditions will appear necessitating a surgical revision. I have seen too often the statement in the records, "enough of the floor was removed to obtain drainage," or "the posterior ethmoid cells were allowed to remain as they did not appear to be grossly infected." The sphenoid sinus is frequently omitted entirely in the operative notes. In the opinion of some of the surgeons, it would seem that "partial" or as is sometimes termed "modified" Lynch operations are justifiable. I cannot agree with this opinion in view of the facts which I have mentioned above. Whether the operation is of the Lynch type, or obliterative, better results could be obtained if more attention were paid to the complete removal of the floor of the frontal sinus, especially toward its outer lateral margin, and posteriorly where it meets the inner table. A complete ethmoidectomy should be done, and special care taken to remove the orbital extensions of the ethmoid. Finally, the sphenoid should not be neglected.

Postoperative care is absolutely essential. It cannot be too strongly repeated that these patients must be watched frequently and over a period of many months to be sure that granulations and adhesions do not form to block drainage. It should be impressed upon every patient to report immediately to the surgeon when any sign of reinfection appears. In hospital services, it would be better for the patient to have regular check-ups by the surgeon who operated upon him rather than to delegate this to interns who have not first-hand familiarity with the case.

SUMMARY

In conclusion, I wish to call attention to:

1. The high percentage of second operations which have occurred in this series.

2. The frequency with which the following three conditions were found at reoperation: (a) scar tissue, (b) remnants of the frontal sinus floor, and (c) ethmoid extensions.

3. No matter whether the operation intends to obliterate the sinus or to provide drainage into the nose by means of a Killian or Lynch type operation, consideration must be given to a complete removal of the frontal sinus floor and the ethmoid cells.

330 Dartmouth St.

LX

THE CHANGING CONCEPTION OF THE MANAGEMENT OF CHRONIC PROGRESSIVE DEAFNESS*

FREDERICK T. HILL, M.D.

WATERVILLE, ME.

Twenty-five years ago the treatment of so-called catarrhal deafness was usually some form of inflation, based on the theory of negative pressure as the etiological factor. One needs only to have recourse to the out-patient records of that period to verify this statement. Tonsils and adenoids were removed and certain intranasal operations performed but largely with the idea of overcoming tubal obstruction. Inflation had been a method of treatment for many years. History[1] tells us that in 1724 Guyot, postmaster at Versailles, catheterized the eustachian tube through the mouth in efforts to improve hearing, and that in 1741 Cleland performed catheterization through the nose. During the 80's and 90's various solutions and medicated vapors customarily were introduced into the middle ear by this means, although Politzer stated that the beneficial effect was due mainly to the inflation of air. Pneumatic massage or the pressure sound of Lucae was used when there was diminished mobility of the drum. Bougies were employed for the treatment of strictures of the eustachian tube.

Then came the era of focal infections and the possibility of toxic absorption causing deafness began to displace the negative pressure theory. Influenced no little by the several publications of Emerson,[2] who took the stand that perception deafness was the result of deep infection in the lymphoid tissue, emphasis gradually became concentrated upon eradication of all possible foci and local treatment was more or less relegated to the background. While the results following the removal of infected foci were satisfactory in certain cases which had not as yet undergone significant degenerative changes, too often these efforts were disappointing, when the initial enthusiasm and the wishful thinking had abated. The eradication of foci of infection seemed more important in the preventive aspect and of less value in the treatment of well-established conditions.

---

*Presented before the meeting of the American Laryngological, Rhinological and Otological Society, Atlantic City, June 1, 1942.

Roentgen therapy of deafness aroused some interest in the early 20's but the claims of Stokes for this method of treatment were not substantiated. In 1926 the late D. Campbell Smythe[3] reported a series of 100 cases of chronic deafness treated by X-ray, concluding that treatment seemed beneficial only in cases of short duration and that improvement was probably due to destruction of lymphoid tissue in the eustachian tube, perhaps a forerunner of Crowe's work of recent years.

Endocrine imbalance and nutritional deficiencies began to receive attention as possible causes of deafness. Drury, who had the advantage of working with Allen Winter Rowe, published several papers on the value of endocrine treatment. Stucky early emphasized the importance of the nutritional factor, basing his conclusions on clinical observations among the Kentucky mountain folk.

Meanwhile the treatment for otosclerosis consisted largely of advising the patient as to readjustment to future deafness. The probable detrimental effect of local treatment and the disappointing results of the internal administration of iodides, and later phosphorus, precluded any other course. While the researches carried on under the auspices of the American Otological Society brought forth much valuable information regarding the embryological aspects and the pathology of otosclerosis, little or no contribution was made to the treatment. Gray[4] suggested the injection of thyroxin into the middle ear but this procedure was short-lived. Later, Mortimer and others,[5] feeling that there was some relationship between atrophic rhinitis and otosclerosis advocated the use of estrogenic substances in the treatment of what he termed "constitutional deafness."

With the discovery of an ever-increasing series of vitamins a great deal of interest became manifest in the nutritional phase of this problem. Covell's[6] animal experiments have shown that vitamin deficiency causes degeneration of the cochlear nerve but it is debatable whether similar conditions occur clinically in man. Jones[7] stated that vitamin therapy is effective if the ganglion cells are viable, provided the hearing impairment is due to vitamin deficiency. Guggenheim[8] suggested a possible regeneration of damaged neural mechanism by calcium, phosphorus and vitamin therapy, and advocated the use of vitamin B in cases of mumps, measles, scarlet fever and typhoid, to prevent nerve deafness. Selfridge,[9, 10] who has done a vast amount of work with vitamin, endocrine and estrogenic therapy, feels that deafness is related to all factors involved in growth, and that, as nutritional deficiencies at the period of gestation may result in later deafness, treatment should be prenatal and con-

tinued through adolescence. On the other hand, Shambaugh[11] made the pertinent observation that, as impaired hearing is not a symptom of the common deficiency diseases, such as pellagra, beriberi or scurvy, it would be doubtful if a lesser avitaminosis would have any selective action on the auditory nerve. Perhaps the disappointing results from this form of therapy may be due to faulty application but it is more probable that, as with focal infections, the greatest value of the biochemical approach is in the field of prevention.

Crowe's[12] work in radiation of nasopharyngeal lymphoid tissue in the prevention and treatment of hearing defects in children reverts somewhat to the negative pressure theory. Lymphoid hyperplasia, in and about the eustachian tube, may cause pathological changes in the middle ear. Crowe and his associates have shown that these result in losses for the higher tones, first manifested in the frequencies well above those for ordinary speech. This indicates the importance of routine testing for these higher frequencies and suggests the value of the monochord for this purpose. Encouraging results have been reported following early radon treatment with Crowe's technic.

For many years surgical efforts to circumvent obstruction to sound waves reaching the internal ear have interested otologists. In 1801 Cooper advocated myringotomy for deafness due to tubal obstruction. This procedure was soon discarded, only to be revived 50 years later, again with disappointing results. The otologists of that period were chiefly concerned with the difficulty of maintaining an artificial perforation of the drum, and various measures such as excision of part of the membrane, excision of the lower part of the handle of the malleus, crucial incision with the application of caustics and puncture with galvanocautery were advocated. During the 70's there was a revival of interest in surgical efforts to overcome obstruction in the conducting apparatus. Division of the posterior fold of the drum membrane and tenotomy of the tensor tympani were practiced by otologists of that period. Schwartz in 1873 proposed the removal of the drum and malleus, and shortly after many otologists were performing ossiculectomies in efforts to improve hearing. Dench,[13] as late as 1902, while admitting much disappointment, felt that this was due to improper selection of patients and reported 76 out of 88 such cases improved in hearing by this procedure. Politzer[14] suggested ossiculectomy to improve hearing when there were marked adhesions in the middle ear, rigid calcification of the drum, ankylosis of the malleus and incus, or permanent stenosis of the tube. Kessel, in 1876, had advocated the removal of the foot plate of the

stapes in otosclerosis. In 1879 Passow trephined the labyrinth through the promontory, a forerunner of later procedures. For a time synechotomy of the crura and extraction of the stapes were practiced, both in this country and abroad. But, Blake, after observing many of these cases, condemned these procedures as both futile and dangerous, and otological surgery for the relief of deafness fell into the discard.

Then, in 1910, Barany made a fistula into the posterior semicircular canal but the improvement in hearing was of brief duration. Four years later Jenkins opened the horizontal canal but again the results were disappointing. Holmgren's work was perhaps the first to attrack popular attention. Later Sourdille brought out his three-stage operation. In this country Lempert developed the technic which has been productive of the best results. Lempert, Campbell, Shambaugh and others doing this work feel that serviceable hearing can be obtained and retained in properly selected cases, if the fenestra remains patent. Lempert[15] claims his new technic, with the fenestra made in the dome of the vestibule, offers many natural advantages. No one can dispute the value of his contributions to technic. One can doubt his claim that operation checks further progress of the condition.[16] Otosclerosis does not progress evenly. Hearing impairment may remain stationary for a long time and then nerve degeneration may result in marked deafness.

All of these procedures, from 1801 to the present, have had as their objective the circumventing of obstruction in the middle ear. The one exception seems to be Hughson's operation in which a graft is employed to eliminate the damping effect of the round window. Recent experimental work of Kobrak, Lindsay and Perlman[17] makes one doubt the efficacy of Hughson's procedure in cases of stapes fixation. They found that when both the oval and round windows were closed with plaster of Paris no response could be obtained either by air or bone conduction. All of these procedures have been in the nature of orthopedic otology, ignoring efforts to remove the underlying pathlogy. It is rather unfortunate that, during all these years, so much more attention has been devoted to operative technic, as compared to physiological research. It seems unfortunate that histologic studies such as Engstrom's could not have come earlier. Perhaps this is only natural because of the urgent demand to do something for deafness. Therapy often does not wait for a physiological basis. Frequently this results in waste of efforts and, on the other hand, sometimes a theoretical premise is later substantiated.

The researches of Bast, Wever and Bray, Davis and Lurie, Fletcher, Wegel, Lorente De No, Crowe, Guild and others, during the past few years, have given us a better understanding of the physiology of hearing and the pathology of deafness. We have gained a better conception of cochlear function and how it may be damaged by acoustic trauma, and by drugs (Taylor, Mosher). The development of the audiometer has given us a more precise means of testing hearing and of evaluating the efficacy of treatment than was possible in earlier years. Had uniformly standardized precision methods been available, undoubtedly much wasted effort could have been avoided.

What then is our present conception of treatment of chronic progressive deafness? Adequate treatment is dependent upon correct diagnosis. This frequently requires considerable study and observation. If we are quite honest with ourselves we might admit that oftentimes the greatest obstacle to correct diagnosis is a busy practice, the demands of which may make difficult the devotion of time necessary for the study of the rather undramatic case of impaired hearing. We will consider the patient's age and occupation, with the possibility of acoustic trauma from industrial noises. We will keep in mind the question of constitutional disease, blood dyscrasia, or endocrine imbalance, remembering that syphilis, leucemia, myxedema or brain tumor, for example, may be seen by the otologist, because of the presenting symptoms of deafness. All of this may demand the cooperation of consultants in other fields of medicine.

A comprehensive history and careful examination should give a good conception of the otological condition, its functional status and some idea of its probable etiology. When there are definite indications of tubal obstruction, usually in cases of short duration, we may employ the eustachian catheter, and sometimes the bougie. But we will not, if we are conscientious, use inflation empirically and ad infinitum, until the patient, discouraged in spirit and poorer in purse, either gives up in despair, or seeks more intelligent treatment elsewhere. The treatment of deafness is the treatment of its cause, and the cause of tubal obstruction will be found, usually in the sinuses or the nasopharynx. Recognition of probable factors such as lymphoid hypertrophy, sinusitis, enlarged posterior tips of inferior turbinates, nasopharyngeal cysts or tumors, or allergic manifestations should lead to more effective therapy.

We are gradually coming to realize that a drop in hearing for the higher frequencies is characteristic of tubal obstruction. It is

very probable that many of our diagnoses of nerve deafness due to toxic absorption, which were based upon this type of hearing loss, were incorrect and that, in the past, undue emphasis has been placed upon focal infection. Loss of bone conduction would seem necessary to substantiate a diagnosis of nerve deafness.

I think we have developed a saner conception of focal infections as causative factors in deafness. We do not see quite as much indiscriminate removal of every removable organ in the nose and throat, or such wholesale extraction of teeth, as once threatened. We have learned that the eradication of foci is of more value in the prevention of hearing impairment than in effecting improvement. If the impairment seems due to a focal infection we will consider all possible foci, not condemning those easily seen in our own field while neglecting others which may be quite as significant. How many patients with nerve deafness due to some such condition as mumps have had tonsils removed when, in all probability, the only indication was the presence of tonsils? How often may some foci in the prostate or the gallbladder be overlooked when attention is concentrated on the nose and throat? Removal of foci is justified when the history suggests a probable relationship, when examination indicates a definite infection, and the hearing status and age of the patient gives a reasonable prognosis for improvement.

What of the so-called medical treatment of deafness? Every now and again some particular drug is suggested, acclaimed for a time and then forgotten. Generally these measures are based upon theoretical premises alone. There is a sort of self-hypnotism among deafened people, who, above all, desire to hear better, which causes them to feel improved, temporarily, under any new form of treatment. Careful testing may show some slight improvement in the audiogram. But repeated testing, through experience, usually allows the patient to respond better, thus seeming to improve. This imagined improvement, plus the natural enthusiasm of the discoverer of the treatment, sometimes leads to unwarranted claims for its efficacy, and it may have rather widespread use until the careful observations of impartial investigators prove its ineffectiveness.

Deafness is a symptom, not a disease, and to treat a symptom empirically with a particular drug, vitamin, or endocrine substance is not the best medicine. If careful study suggests some endocrine or vitamin deficiency, indicated treatment should be carried out, but with the idea of correcting such deficiency and improving general health rather than for any specific effect upon the auditory nerve.

Radiation therapy, in doses sufficient to be effective, may be a two-edged sword and, in its present state, does not seem advisable for indiscriminate employment. It is still somewhat experimental and its application can well be left to those having the facilities for adequate treatment and the experience to govern its use.

It would seem that, after 140 years, the operative treatment of deafness at last had been improved to the point of practicability. Lempert[15] reports no tendency to closure of the fenestra in the 75 patients operated upon by his new technic. Those experienced in this work feel that this operation offers serviceable hearing in cases of deafness due to stapes fixation, in which there is good cochlear function for the conversational frequencies, as determined by bone conduction. Many of us, recalling the enthusiasm evoked by similar efforts in the past, will be inclined to await the results of further observations before considering the problem of permanent fenestration settled beyond question of doubt. We agree with Kopetzky's[17] statement that it is impossible to estimate definitely the value of fenestration to the patient but that this type of surgery should be continued. In certain well-selected cases of binaural deafness due to stapes fixation this operation may be justified, if the patient thoroughly understands the present status of this type of surgery.

In the majority of cases the use of a properly selected hearing aid is the best and most practical method of overcoming the handicap of an established deafness. Marked improvements in recent years, notably in the development of the vacuum tube type of aid, and the recognition by the otologist of his responsibility for seeing that his patient gets the aid best suited to him, have made this form of therapy more effective than was formerly possible.

Perhaps the most potent single influence in the prevention of deafness and the conservation of hearing has been the educational campaign carried on by the American Society for the Hard of Hearing, aided by a number of interested otologists. This has stressed the importance of early recognition of hearing defects, the need for careful audiometric tests for all school children, proper medical follow-up for those found deficient, and remedial measures, such as instruction in lip-reading and vocational guidance, for those requiring it. Dr. Horace Newhart,[19] Chairman of the Committee on the Prevention and Amelioration of Deafness of the American Academy of Ophthalmology and Otolaryngology, has recently added another objective; to protect workers in industry from the effect of noises detrimental to hearing. The importance of this can hardly be overestimated.

SUMMARY

We realize the impossibility of curing deafness. All of our therapeutic efforts finally come down to prevention; correction by circumvention, or amplification; or rehabilitation. The changing conception of the management of chronic progressive deafness, through these years, is a result of better understanding of the physiology of hearing and the pathology of deafness, and the development of precision methods of testing. We retain many of the older procedures, indeed have revived some of those discarded, as their once theoretical premises have been substantiated by modern research. We recognize the effect of tubal obstruction, of negative pressure, and try to remedy its cause, either by local treatment, surgical measures in the nose or throat or by irradiation. We keep in mind the possibility of toxic absorption and strive to eradicate foci of infection when indicated. We consider the effect of occupation or environment. We realize the importance of good general health and advise the correction of any nutritional or endocrine deficiency. And above all we urge the need for early recognition and correct diagnosis, if prevention is to be effective. For the majority of our patients with established deafness, sufficient to be handicapping, correction by amplification will be preferable. Operative treatment may be suggested to those who, after critical study, meet all the requirements laid down as essential to successful fenestration, provided they fully understand its present status. And finally we recognize the importance of cultivating a personal interest in each patient, adapting treatment to individual circumstances and conditions, and endeavoring in all possible ways to assist him to achieve an improved social and economic life.

PROFESSIONAL BLDG.

REFERENCES

1. Robinson, V.: Chronology of Otology, Bull. Hist. Med. 2:199 (July), 1941.

2. Emerson, F. P.: Perception Deafness, Tr. Amer. Otol. Soc. 15:339, 1921.

3. Smythe, D. C.: Treatment of Lateral Pharyngeal Bands for Deafness by X-ray. Result of 100 Cases, ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY, 35:1157 (Dec.), 1926.

4. Gray, A. A.: Treatment of Otosclerotic and Similar Types of Deafness by Local Application of Thyroxine, Laryngoscope 45:741 (Oct.), 1935.

5. Mortimer, H., Wright, R. P., Thomson, D. L., and Collip, J. P.: Intranasal Administration of Estrogenic Hormones in Constitutional Deafness, Canad. M. A. J. 40:17 (Jan.), 1939.

6. Covell, W. P.: Pathologic Change in Peripheral Auditory Mechanism Due to Avitaminosis, Laryngoscope 50:632 (July), 1940.

7. Jones, I. H.: Vitamin and the Ear, Nose and Throat, Laryngoscope 51:609 (July), 1941.

8. Guggenheim, L. K.: Treatment of Deafness, Laryngoscope 50:773 (Aug.), 1940.

9. Selfridge, G.: Present Status of Vitamin in Relation to Eighth Nerve and Conduction Deafness, Arch. Otolaryng. 34:125 (July), 1941.

10. Selfridge, G.: Chronic Progressive Deafness With Special Reference to Estrogenic Substances, ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY, 49:53 (March), 1940.

11. Shambaugh, G. E., Jr.: Chronic Progressive Deafness, Progress in Otolaryngology, Summaries, Arch. Otolaryng. 33:436 (March), 1941.

12. Crowe, S. J.: Recognition, Treatment and Prevention of Hearing Impairment in Children, Laryngoscope 50:658 (July), 1940.

13. Dench, E. B.: Ossiculectomy, Med. News, N. Y., Feb. 28, 1903.

14. Politzer, A.: Diseases of the Ear, Philadelphia, 1926, Lea & Febiger.

15. Lempert, J.: Fenestra Nov-ovalis, Arch. Otolaryng. 34:880 (Nov.), 1941.

16. Lempert, J.: Endaural Fenestration of Horizontal Canal for Otosclerosis, Laryngoscope 51:330 (April), 1941.

17. Kobrak, H. G., Lindsay, J. R., and Perlman, A. B.: The Next Step in Auditory Research, Arch. Otolaryng. 31:467 (March), 1940.

18. Kopetzky, S. J.: History and Present Status of Operations on the Labyrinthine Capsule for Otosclerosis, Surg., Gynec. & Obst. 72:466 (Feb.), 1941.

19. Newhart, H.: Progress in the Conservation of Hearing, ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY, 50:129 (March), 1941.

LXI

DERMOID CYSTS OF THE NOSE*

EDGAR M. HOLMES, M.D.

BOSTON

The literature of the past has presented but a few cases of dermoid cyst of the nose. There have been approximately 29 articles in the literature on the subject and only five of these are of American origin. Since the author has had two cases within a month and has seen three others within the same year, a review has been made of the cases recorded at the Massachusetts Eye and Ear Infirmary for the past twenty years.

Dermoid cysts of the nose are due to faulty development of the nasal structures during embryonic life. It is assumed that the frontal plate of the embryo which forms the nose is composed of hyaline cartilage covered externally with epidermis and internally with a mucosa. As stated by Luongo,[4] the bony tissue after the third month extends in between the skin and cartilage. This bone will form the nasal bones and the cartilage becomes absorbed during the process. During the gradual separation of the skin from the cartilage of the frontal nasal plate by the intrusion of the nasal bones, a small portion of the skin or epithelium sequestrates and develops into a dermoid cyst.

Benjamin[1] believes that the cysts result from faulty development in the region of the sulcus supranasalis and are the cause of the defect which occurs in the vicinity of the glabella.

Dermoid cysts may be present and conspicuous at birth but more commonly they do not reveal themselves until later on in life. The most common age at which this group of cases were first observed was from 10 to 20 years. The reason for this is due to their microscopic structure. The walls of these cysts are usually comparatively thick and are lined with a squamous epithelium which contains hair follicles, sweat and sebaceous glands. Thus we have present all the elements of the normal skin. At birth these inclusion elements are very small and may be microscopic but during

*From the Department of Otolaryngology of the Massachusetts Eye and Ear Infirmary.

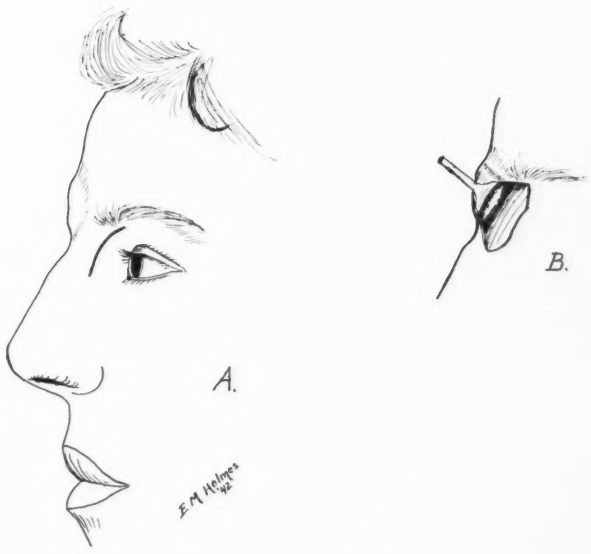


Fig. 1.—*A*. The location and amount of swelling of the dermoid cyst at the glabella. The incision for the exposure is designated by the heavy line half-way between the dorsum and the eye. *B*. The exposure obtained by a long-bladed spatula. The cyst was in reality a flattened disc with a stalk that went into a depression in the frontal bone near its center.

growth the products which normally grow to the surface of the skin, the hairs, desquamated epithelium, sebaceous and sweat secretions, are entrapped within the center of the mass. This accumulation forms a core about which the epithelium proliferates and stretches, becoming larger and larger.

It is following this period of gradual accumulation that the tumor presents itself. These cysts may occur at any line of fusion of the embryo. Thus they are seen not only in the nose but about the auricle, the orbit, the floor of the mouth, the anterior fontanel and the occipital protuberance.

Dermoid cysts of the nose usually start in connection with either the nasal or frontal bones so that even if the cyst presents itself at the tip of the nose, its stalk of origin will be found to extend to and be in contact with either the nasal or frontal bones. In reviewing

the cases treated at the Massachusetts Eye and Ear Infirmary during the past twenty years, both private and ward cases have been included. There have been 15 patients, all of whom were operated upon.

The swelling presented itself at the glabella in five cases. In the rest the swelling was divided between the tip of the nose and along the dorsum about the tip of the nasal bones. Five cases were predominantly at the tip; the remaining five being along the dorsum. In all of this latter group it was found that the cyst extended to the tip of the nasal bones usually going beneath them and in most of the cases creating a depression or a definite sac within the septum.

Three cases are presented not as interesting case histories, as most of them follow a common pattern, but as operative problems.

Taken as a whole these cases presented a swelling of the nose which had been observed some time following the birth of the child. In some the swelling remained encapsulated and was retained beneath the skin. In others the sac had become infected and broken through the surface creating a draining fistula. When this was the case, the attending physicians had invariably incised the mass, curetted it, applied some caustic to destroy the lining epithelium and allowed the defect to close by second intention. The majority of their results were discouraging as the lining of the cysts had not been destroyed completely. Even a trace of remaining germinal epithelial tissue or gland structure is sufficient to recreate a new cyst. It therefore is imperative to eradicate the cyst completely or establish a large drainage fistula which will carry away the products of proliferation.

The first case is that of a 13-year-old girl who had a swelling in the midline between the brows at the glabella. It was flat, nonadherent to the skin, and yet was apparently adherent to the bone. X-rays revealed a slight depression in the bone at its point of attachment in the frontal bone (Fig. 1).

Simple excision of the cyst by a transverse incision over the mass would have been the easiest approach but would have left a moderately prominent scar on the dorsum of the nose at the glabella where it would have been most conspicuous. To avoid this an incision was made on the side of the nose about midway between the inner canthus and the dorsum which would correspond to an incision made for an exposure of the ethmoid. Through this incision the dissection was carried out, the sac exposed, its termination followed to a depression in the bone which corresponded to that seen in the

x-ray. The stalk disappeared as fibrous strands going into the bone. Following the removal of the sac which came out intact, the skin incision was closed with interrupted atraumatic plastic sutures and the cavity created beneath the skin by the removal of the sac was obliterated by a pressure bandage. Two weeks following the operation there was no evidence of a depression at the site of the removal of the cyst and the scar on the side of the nose was barely visible.

The second case was that of a young man 18 years of age who had had a fistulous tract on the dorsum of the nose below the tip of the nasal bones incised, curetted, and cauterized on two previous occasions by other operators. Examination revealed a broad scar on the dorsum extending from the end of the nasal bones half-way to the tip of the nose. In the center of this was a fistulous tract going up to and slightly beneath the nasal bones. Our problem here was to excise not only the sac which was not apparent, but the broad scar over the dorsum of the nose so that when the edges were approximated there would be as little scarring as possible. The other problem was to remove the sac completely beneath the tip of the nasal bones. It seemed probable that this would require removal of some of the bone. The procedure employed was the following (Fig. 2):

The scar tissue which had resulted from previous infections, excisions and cauterizations was completely excised. The normal subcutaneous tissues lateral to the scar were then exposed. There were the nasal bones, the lateral and alar cartilages. Working medially from normal tissue, the cyst was found to lie in a trough created by a lateral and downward displacement of the lateral cartilages and a depression in the septal cartilage. The cyst was easily dissected out of this and its pedicle was found to go in under the nasal bones. As the pedicle was firmly attached to the nasal bone and the exact depth to which the lining went could not be determined, a wedge from the tip of the bones was excised including the attached cyst. This created a broad as well as depressed dorsum and an opening into the nasal cavity. In view of the fact that the patient had a slight hump, a rhinoplastic procedure was employed to create a pleasing dorsum and at the same time narrow the nasal arch. More nasal bone was removed from the dorsum and sufficient septal and lateral cartilage was excised to create a straight dorsum. A wedge of bone was then removed between the septum and each nasal bone so that the dorsum could be narrowed as high as possible. The nasal bone and part of the ascending process of the superior maxilla were then separated with a chisel and the dorsal margins brought to the midline. The free cut margins of the lateral cartilages were sutured together and the skin

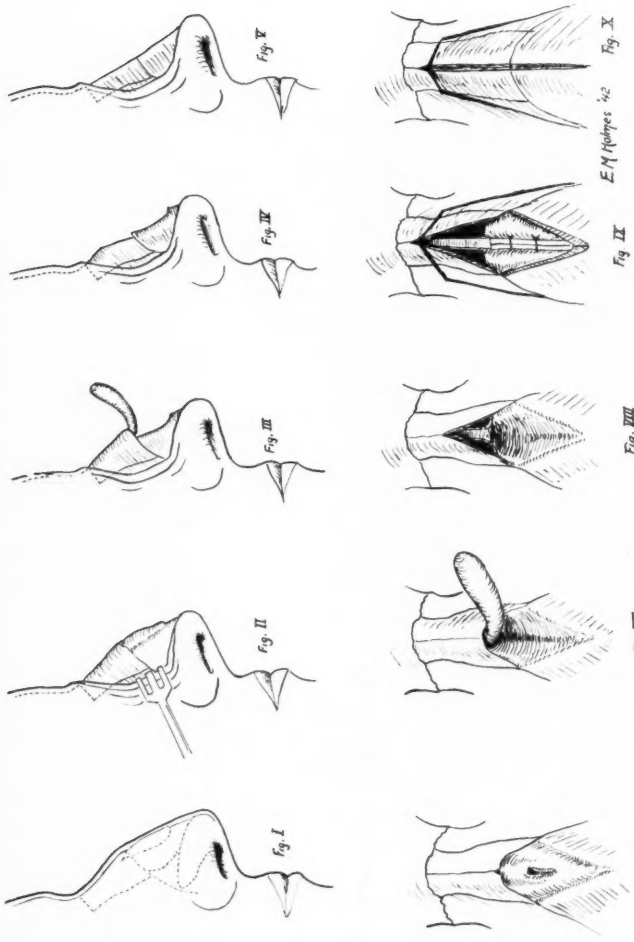


Fig. V
Fig. IV
Fig. III
Fig. II
Fig. I
Fig. X
E.M. Holmes 1/2
Fig. IX
Fig. VIII
Fig. VII
Fig. VI

Figs. 2.

I.—Schematic drawing portraying the cyst in relation to the skin, nasal bones and lateral cartilages.

II.—The skin retracted laterally following an incision outside the scarred cystic area.

III.—The cyst has been elevated from its bed showing it adherent to and extending beneath the tip of the nasal bones.

IV.—The cyst, together with the tip of the nasal bones, has been removed leaving an irregular dorsal ridge.

V.—The contour of the nose has been straightened following the removal of further bone and cartilage.

VI.—The nasal bones, cartilages and cyst are shown following the removal of the skin. There was a depression in the center of the cyst through which a probe could be inserted upward under the nasal bones.

VII.—The cyst is here lifted from its bed which consisted of depressed and broadened septal cartilage. The lateral cartilages apparently had not approached the midline at this point.

VIII.—Following the removal of the cyst with the stalk and the tip of the nasal bones, the cavities were entered and normal septum encountered.

IX.—This view shows three points. More nasal bone has been removed together with a wedge of solid bone between one another at the apex. The septal cartilage which had been flattened into a depression and fanned out was cut along the margin of the depression; separated from the nasal mucosa; hinged at the septum with their free ends brought to the midline where they were sutured. This filled in a depression at this point. The dark straight lines show the line of fracture of the nasal bone and ascending processes of the superior maxilla.

X.—The nasal bones and lateral cartilages have been brought to the midline.

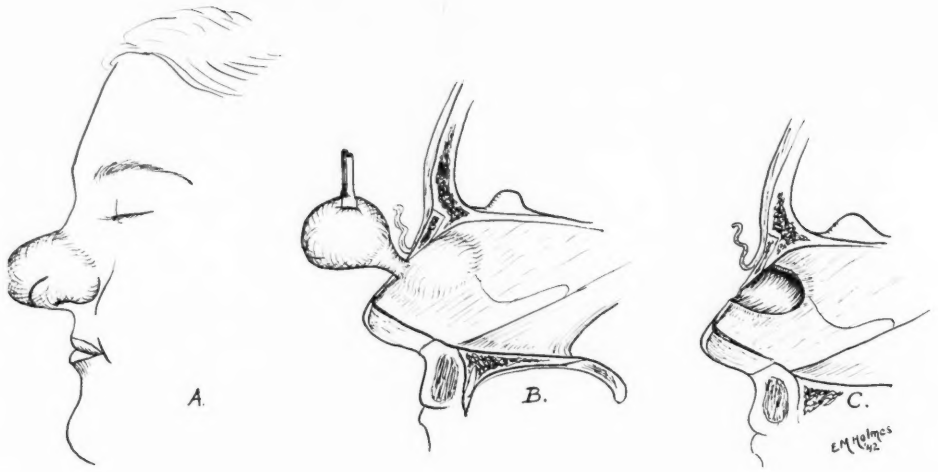


Fig. 3.—*A*. The size and location of the cyst. The butterfly incision for exposing the cyst is shown by the darkline on the under surface of the tip. *B*. In this schematic drawing the main part of the cyst that protruded outside the nose is held up. The stalk that penetrates the center of the dorsum beneath the tip of the nasal bones is portrayed and the remaining cystic swelling between the flaps of mucosa in the septum is demonstrated. *C*. The lateral wall of the sac within the septum has been excised, as much of the cystic wall membrane removed as was possible and the cavity left open into the nose.

was approximated with interrupted atraumatic sutures following which a pressure bandage was applied. Two weeks following the operation the incision was healed and there was little reaction about the operative field. Two months later there was only a moderately visible midline scar.

The third case was one of Dr. V. H. Kazanjian's. The patient was a girl 14 years of age whose nose had a very large, bulbous, inflamed tip which had enlarged since childhood. Examination revealed a cystic, round, shiny, inflamed, tense mass involving the tip of the nose. The swelling of the tip was 1½ inches in diameter. Intranasally there was visible a swelling of the septum, high and anteriorly. (Fig. 3.)

In operating on this patient Dr. Kazanjian made a butterfly incision in the tip of the nose. The sac was then dissected out and found to be quite thin-walled. There was displacement of the alar cartilages laterally as well as downward and they were also thinned out. The dorsum of the nose was slightly depressed up to the nasal bones, which were normal. At the nasal bones the sac penetrated the septum in the midline and was about ¼ inch wide at that point. On attempting to dissect this out, it was found that there was a large dilation and sac in the septum high up in under the nasal bones. This sac he found impossible to remove completely because of its extent and in order to prevent recurrence of the cyst, he employed the simple procedure of creating a large communication between the sac and the interior of the nose by removing one entire side of the cyst. Thus all of the external cyst was removed and the remaining lining was left exposed within the nose so that any epithelium, hairs or sebaceous material would exfoliate into the nasal cavity where they would be automatically taken care of by nature or could be removed manually. The exterior of the nose was then closed after excess abnormal skin had been excised. The cartilages were brought into place and the resulting nose was very satisfactory.

The majority of the other cases consisted of simple excisions of a cyst preceded by an incision over the mass.

SUMMARY

A swelling within or beneath the skin of the nose if not an abscess or sebaceous cyst may very likely turn out to be a dermoid cyst. One must always be on the alert for a meningocele. Dermoid cysts must be completely removed before a cure can be obtained as the slightest vestige of remaining cyst wall or glandular cells can

cause the cyst to regenerate. These postoperative cysts inwardly become infected or break through the old incision creating a draining fistula. A brief review of 15 cases of patients operated upon at the Massachusetts Eye and Ear Infirmary in the past twenty years have been reviewed and the operative procedures in three of them discussed.

330 Dartmouth St.

REFERENCES

1. Benjamins, C. E.: Congenital Epithelial Passages of the Nasal Bridge, Acta oto-laryng. 24:284, 1936.

2. Hagens, E. W.: Congenital Dermoid Cyst and Fistula of the Dorsum of the Nose, Arch. Otolaryng. 28:399 (Sept.), 1938.

3. Juers, A. L.: Dermoid Cyst of the Nasal Septum, Arch. Otolaryng. 33:851-52 (May), 1941.

4. Luongo, R. A.: Dermoid Cyst of the Nasal Dorsum, Arch. Otolaryng. 18:755 (June), 1933.

5. Parkinson, S. N.: Dermoid Cyst of the Nasal Bridge, Arch. Otolaryng. 34:829-31 (Oct.), 1941.

LXII

A COOPERATIVE BRONCHOSCOPIC CLINIC

Gordon D. Hoople, M.D.

Syracuse, N. Y.

Prior to 1935, bronchoscopy in Syracuse was at sixes and sevens. This was the fault of no one in particular. There was no guiding genius to organize the work. None of the internists were notably interested in this phase of medical diagnosis and treatment. There were seven physicians, all diplomates of the American Board of Otolaryngology, who could perform bronchoscopies, but only occasionally did one. Certainly the great majority of bronchoscopies were for the extraction of foreign bodies. Each of the seven men possessed his own equipment, and transported it from his office to the particular hospital where he might be asked to work, when he was called upon to bronchoscope some patient. The equipment was necessarily inadequate to meet the needs of a city the size of Syracuse. The infrequency with which bronchoscopies were done was a factor which limited skill and proficiency.

This state of affairs in Syracuse was not due to a late start. Thomas Halsted of this city was one of the pioneers of bronchoscopy in this country. He performed the first bronchoscopies done in Central New York. In these early days he frequently consulted with Chevalier Jackson. At a later date others came to Syracuse to practice the art but no one had especial interest in this branch of medical practice. Individual effort was the order of the day.

It became evident in the early thirties that some effort must be made to place bronchoscopy on a sounder basis. Reports from various clinics throughout the country, apart from Philadelphia, showed that there was a need in every city of some size for better diagnostic work in this field. Results were too conclusive to be ignored. What should be done in Syracuse?

The problems were many. The country had just passed the low point of the great depression. The medical institutions of Syracuse were without funds to help establish a bronchoscopic clinic. The medical college was going through the throes of securing a new building (since obtained) and was in no position to help. The major hospitals of the city had financial troubles of their own. No indi-

vidual was in sight, nor was one discovered, who could furnish financial aid. Because these conditions existed, the problem of housing was serious. The question of staff personnel needed to be solved. It was a discouraging picture.

Dr. Frederick J. O'Connor, who was just beginning his service as head of the Department of Otolaryngology at the College of Medicine of Syracuse University, called a meeting of the men of the department to consider the problem. He suggested to the group that a bronchoscopic clinic be established and that it be staffed by the group assembled. He further suggested that the clinic be housed in the hospital whose arrangements were best suited for the purpose and that each one of the group contribute what instruments he privately owned toward the equipment of the clinic. The members of the department agreed to follow his suggestions.

Consultation was held with the authorities of the Syracuse Memorial Hospital and they voted to grant space in which a weekly meeting of the clinic could be held. This was a fortunate decision for the clinic as the operating suite of this hospital was the largest in the city and really provided ample room. Dr. O'Connor was able to secure a small amount of money for the purchase of some very much needed additional equipment. Only the bare requirements for diagnosis, biopsy and extraction of foreign bodies were in the initial armamentarium. With this inauspicious start the clinic held its first session in the first week of January, 1935.

At the very start the group might have been without clinical material had it not been for the interest of Dr. Joseph R. Wiseman, head of the Department of Allergy in the College of Medicine. His interest was in the field of asthma and his desire to know the possible benefits that bronchoscopy might afford patients afflicted with this condition furnished the clinic with much of its material during the initial years of its operation. Some of these initial patients proved to be suffering from conditions other than asthma. An occasionally unsuspected bronchogenic carcinoma was discovered and other interesting conditions revealed. As these discoveries became known, the scope of the work of the clinic enlarged and soon it was on a much improved basis.

For five years the entire staff attended the weekly meetings (it was semiweekly on some occasions). There was much to be learned and inasmuch as no one of the group had been specially trained in endoscopy, all profited by mutual consultation and observation. A spirit of cooperation was developed through this endeavor which is the envy of other branches of medicine in the city. Nearly

three years ago the work was divided so that it is now carried by four services which run three months each year.

The staff of the clinic at present is made up of representatives from four of the city's six major hospitals. Inclusive and cooperative representation accounts for the fact that almost all of the bronchoscopic work done in Syracuse is done in the clinic quarters with the use of the clinic instruments. This applies both to ward and private cases. If one of the four hospitals mentioned above has on its medical service a patient for whom bronchoscopic examination is desired, that patient is transferred to the Memorial Hospital, together with X-rays and pertinent data. Here the patient is bronchoscoped and then transferred back to the referring hospital, with a report of the bronchoscopic findings. An otolaryngologist on the staff of the referring hospital may or may not be present. If one is on service at the moment he will, of course, be in attendance, but it frequently happens that the work will be done by a member of the staff of one of the other participating hospitals.

This method of operation may appear cumbersome and unwieldy. It has some disadvantages to be sure, but it is felt that the good points outweigh these. First and foremost, this setup has made for better bronchoscopy. There is no question about this. True, there have been mistakes in judgment and in execution, but they have been fewer and less serious than they might have been if there had been four operating units instead of one. Second, the cost of equipment to the hospitals and the doctors, and thus to the citizens, has been low. Third, the cost of running expenses and the cost of maintenance has been materially less. In these days of high medical costs this is a factor which has merit.

The clinic has been fortunate in another respect. The Nursing School of the Memorial Hospital, early in this work, felt the need of properly trained nursing personnel for the direction of the bronchoscopic room. Arrangements were made with the Nursing School of the Jefferson Hospital in Philadelphia, so that a nurse could have a period of short affiliation for the purpose of learning bronchoscopic nursing technique. During the past seven years there have been some changes in nursing personnel. A period of observation and training has been extended to each new supervising nurse. This has been of distinct advantage to the physicians in the clinic in the past. The cooperating bronchoscopists desire a continuation of this happy relationship.

A study of the statistics of the clinic (Table I) reveals a growth in the amount of work done, which is apparently healthy.

TABLE I

NUMBER OF PROCEDURES

Year	Bronchscopy	Esophagoscopy	Laryngoscopy	Total
1935	160	18	4	182
1936	112	19	30	161
1937	139	19	27	196
1938	121	50	37	208
1939	101	43	40	184
1940	168	90	60	318
1941	170	99	49	318
			Total,	1567

A condensed classification according to diagnosis of the work done in the three divisions (Table II) brings out a few interesting facts.

TABLE II

	Bronchoscopy	Esophagoscopy	Laryngoscopy
Tumors, Benign	9	7	33
Tumors, malignant	24	14	29
Foreign bodies	27	90	14
Lung abscess	32		
Bronchitis	32		
Asthma	68		
Bronchiectasis	41		
Atelectasis	9		
Tuberculosis	20		
Miscellaneous	14	15	15
Diagnosis undetermined	82	15	10
Strictures		17	
Cardiospasm		10	
Laryngitis and laryngotracheobronchitis			41
Undeveloped, congenital			17
Paralysis			6

To the doctors of Syracuse and its environs, biopsy of tumors for the establishment of a definite diagnosis has been perhaps the most helpful feature of the clinic. Many of the malignant tumors of the bronchi were undiagnosed and some unsuspected before bronchoscopy.

Foreign body cases comprise about 8 per cent of the total which approximates the oft-suggested figure of 5 per cent for clinics of this kind.

Cases of undetermined diagnosis are greater in number than we would like. This group, however, includes those with some pathologic condition which cannot be diagnosed by endoscopic means. An example would be a patient with some kind of mediastinal tumor, with negative bronchoscopic findings.

SUMMARY

This presentation tells of the origin and growth of a cooperative bronchoscopic endeavor in a city of approximately 200,000. The work of this clinic is done in one of four cooperating hospitals. The staff is made up of members of the Department of Otolaryngology of the College of Medicine of Syracuse University. The work is done by members of the staff though each may be a member of the staff of only one or two of the four cooperating hospitals.

Such a cooperative setup would not be practical in large metropolitan centers. Distances between hospitals and the amount of work to be done would be deterring factors. In small communities with but one hospital, it would not be possible. However, there must be several localities where a similar enterprise would meet needs not now met, reduce overhead cost, improve endoscopic work in the community, and create medical fraternity where there is now unnecessary rivalry.

MEDICAL ARTS BLDG.

LXIII

MÉNIÈRE'S DISEASE*

CHARLES I. JOHNSON, M.D.

BOSTON

In 1861 Ménière read a paper before the Academy of Medicine in Paris on a syndrome often mistaken, up to that time, for cerebral apoplexy. He reported 11 cases with one necropsy. The post-mortem gross examination on his one case showed hemorrhagic exudate in the semicircular canals with the brain cerebellum and spinal cord free of changes. He was wrong in his conception of the pathology in these cases, thinking they were all due to hemorrhage into the labyrinth.

However, he definitely made an epochal advance toward exact diagnosis by placing the pathology in the peripheral vestibulo-cochlear apparatus and saved many patients from the bleeding, purging, sweating, and other heroic measures employed in treating cerebral apoplexy. He recognized the fact and admitted that intracranial lesions could give rise to similar phenomena.

With the development of Bárány tests as a means of studying vertigo and our increasing knowledge of labyrinthitis, all became convinced that the pathology of Ménière's syndrome was in the peripheral vestibulo-cochlear apparatus where Ménière had placed it.

We know today that equilibrium is maintained by tonic impulses, emitted constantly from both labyrinths, which act synergistically. Each labyrinth initiates reactions in gait, and in eye, body and limb positions in opposing directions but with unequal force, this inequality being in the proportion of two to one and the prevailing force operating to the opposite side. Thus, the right labyrinth causes the body, limbs and eyes to deviate to the left with a force twice as great as to the right. The left labyrinth causes similar deviations to the right with a force twice as great as to the left. In health and at rest these reactions cancel each other and a state of equilibrium exists.

---

*From the Department of Otolaryngology of the Massachusetts Eye and Ear Infirmary.

Interference with the peripheral mechanism produces an upset in equilibrium involving all of the following: (1) disturbance of gait and of posture, with staggering and falling; (2) disturbance of limb motion, as in past pointing; (3) eye deviations or nystagmus; (4) a sense of motion or vertigo; (5) vagus phenomena such as pallor, sweating, nausea, vomiting and fainting; (6) disturbances in hearing.

If one labyrinth is completely destroyed, the other one in time takes over the fine coordination of both, but so long as the function of one is in a state of change, coordination is impossible and the syndrome of peripheral disturbance results.

Even with our present-day knowledge it is not easy to make a diagnosis of Ménière's syndrome. It cannot be done satisfactorily without the aid of the internist and the neurologist. We are apt to think of all vertigo as a Ménière's syndrome or one of the pseudotypes.

In a recent paper McNally found that 10 per cent of 600 patients admitted to the Montreal General Hospital complained of vertigo. In 50 patients with high blood pressure, 21 complained of vertigo. In 50 patients with low blood pressure, 10 complained of vertigo. In 20 patients with traumatic shock, two complained of vertigo. He and his medical colleagues believe that a fluctuating blood pressure is more apt to cause vertigo than a high or a low blood pressure. In 90 patients with gallbladder disease, 18 suffered with vertigo. In 50 patients with neuritis, eight had vertigo. In 25 patients with avitaminosis, six had vertigo.

Other mentioned causes of peripheral vertigo were otosclerosis, hemorrhage, acute illness (such as mumps or scarlet fever), trauma from accident or as seen in caisson workers.

Among the neurological causes of vertigo are: (1) brain tumor, which although not an important cause and usually not giving episodic attacks, may produce vertigo; (2) vascular lesions, especially of the posterior inferior cerebellar artery, which produce severe vertigo (in cerebral arteriosclerosis the vertigo is usually transient and fleeting); (3) degenerative lesions, as in multiple sclerosis in which 11 per cent of patients have vertigo as an initial symptom.

Central vertigo is usually nonepisodic, nonsystematized and of longer duration while peripheral vertigo is episodic, clean cut, systematized and of shorter duration.

In pseudo-vertigo there may be faintness or a sensation of darkness or of the ground rising or a rocking to-and-fro. These symptoms may be from the end organ or more likely from cerebral anemia, arteriosclerosis, hypoglycemia, neurasthenia or ocular disturbance.

I have wandered off my subject only to avoid discussing vertigo, or so-called Ménière's syndrome, where a known etiology is established as in focal infection, systemic disease, lack of vitamins, allergy, apoplexy of the inner ear, psychosis, and eustachian tube closure and so on. It is not difficult to see how so delicate and finely balanced an organ could be thrown out of kilter and lose its rhythm by one of many causes.

The original description of the syndrome by Ménière is as follows:

"An auditory apparatus, previously perfectly sound, can become all at once the seat of functional disturbances consisting of noises of various characters continuous or intermittent; and these are soon accompanied by a more or less great diminution of hearing. These functional disturbances having their seat in the internal ear often cause attacks considered cerebral such as vertigo, staggering, uncertain gait, rotations and falling, and are besides accompanied by nausea, vomiting, and a syncopal state. These attacks, which are of intermittent form, are soon followed by deafness more and more grave and often hearing is even suddenly and completely abolished".

It is the Ménière's syndrome of unknown etiology that interests us, where all the possible medical, neurological and otological causes have been ruled out. Besides the very severe types there can be moderately severe and mild forms, where only a few of these symptoms and signs present themselves and to a much less degree but usually in the true labyrinthine pattern.

Because the aural symptoms were sometimes relieved by pilocarpine and because the patients grew worse with an increase in weight and better following a decrease in weight, Mygind and Dederding felt they were dealing with an abnormal water metabolism causing fluid retention in the labyrinth. They used a low sodium diet in order to increase diuresis, limited fluid intake, and added physiotherapeutic measures, and at the same time the patient was hospitalized for a minimum period of two weeks.

Furstenberg felt that in the process of water elimination Mygind and Dederding were also eliminating electrolyte from the body in

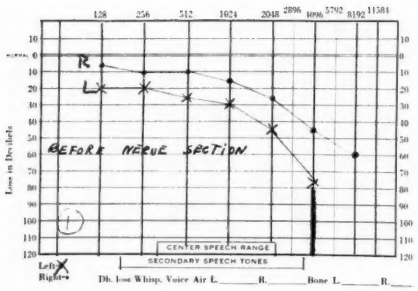
the form of sodium, giving them their good results. He could produce attacks by administering sodium, regardless of the fluid balance. He, therefore, limited sodium intake by his salt-free diet and gave large doses of ammonium chloride to further eliminate and counteract the effect of sodium and obtained excellent results. However, he also found it necessary to hospitalize the patients in order to control their diets and their sodium intake. In our experience few patients have ever been able to tolerate 90 gr. of ammonium chloride per day.

Talbot and Brown, recognizing some efficacy in the treatment of these patients with a low sodium diet, observed that Furstenberg's diet was rich in potassium. They could not reproduce symptoms by alkalosis, hydration or high sodium concentration. Therefore, they paid no attention to the sodium intake but prescribed large doses of potassium chloride to 48 patients and obtained impressive clinical improvements. They feel that the diuretic effect of potassium, which hastens the excretion of water and sodium, is valuable but not the whole story. They suggest that proper conduction of the nerve impulse is found in a medium where optimum concentration of potassium is present.

Everyone bemoans the scarcity of autopsy material in these cases. Halpike has reported the post-mortem findings in two patients who died following section of the eighth nerve. His microscopic sections showed a gross dilatation of the entire endolymph system. Whether it was due to an oversecretion of endolymph or its faulty absorption he could not tell but assumed the latter to be true because of an absence of perisaccular connective tissue.

The chief reason for doubting that all pathology is in the end organ is the fact that after section of the entire eighth nerve, tinnitus may not disappear. Why is not a good simile, the sensations felt in an amputated extremity? After losing a leg the patient may feel pain or sensory changes in the part that has been removed. Scarpa's vestibular ganglion and the spiral ganglion of corti are both distal to the site of auditory nerve section.

The vertigo of Ménière's syndrome is usually sudden in onset and incapacitating but may be mild and atypical. It may last for a few minutes or even days or weeks. The vertigo is frequently characterized by a sense of whirling in the patient himself or in objects about him and aggravated by movements of the head. It may be like the vertigo of the caloric test or labyrinthitis while the labyrinth is dying, accompanied by nausea, vomiting, falling, pallor, sweating or syncope.



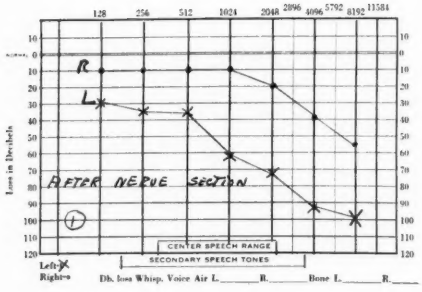


Fig. 1.—Audiogram of hearing before and after operation.

Vertigo was the first symptom in 74 of Crowe's series of 119 patients.

Nystagmus is present during an attack and is usually toward the more normal labyrinth. It is a helpful diagnostic sign when observed.

Deafness is always noted sooner or later. It may come on slowly after repeated attacks and be unilateral or bilateral. If bilateral, the more involved ear is likely the offending one. The deafness is of the inner ear or perception type and is progressive. Deafness was the first symptom in only 14 of Crowe's 119 patients. It may not be complained of by the patient and found only after doing an audiogram.

Tinnitus occurred as a first symptom in 31 of Crowe's series. If it increases in one ear during an attack it is of value in lateralizing the involved side.

Loss of consciousness may occur in 10 to 15 per cent of cases and is very likely due to a drop in blood pressurs.

What can we as otologists do in managing a case? In the first place, it is very necessary that we rule out middle ear disease, such as secretory ears or blocked eustachian tubes.

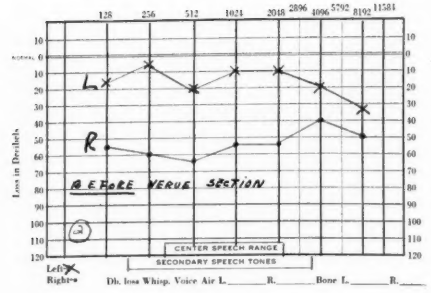
I recall one patient suffering severe vertigo with an hemangioma of the cerebellum and petrous pyramid which presented itself in the middle ear and showed up through an intact drum. The drum was incised, a specimen was obtained, and histologically it proved to be hemangioma.

Cholesteatoma can give severe labyrinthitis, and it may be found in a perfectly dry ear.

Granted that we have a healthy and normal looking middle ear apparatus, an audiogram is in order and invariably it will show a perception deafness.

The Bárány tests usually show a percentage of loss in activity which corresponds roughly with the percentage of hearing loss, but this may vary. All induced phenomena have the normal pattern but are sluggish in response and diminished in degree.

We see very few of the severe incapacitating types of Ménière's syndrome. In the milder forms I have had excellent results with potassium chloride.



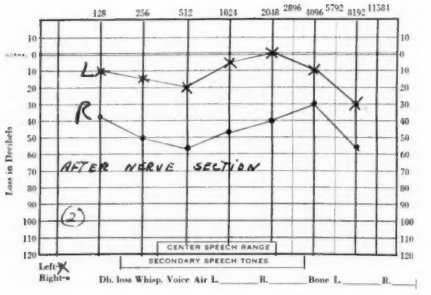


Fig. 2.—Audiogram of hearing before and after operation.

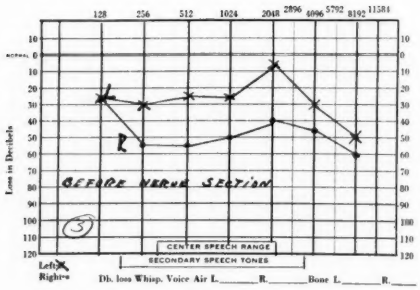
In trying to find patients in our private practices on whom a nerve section had been done, I found Dr. LeRoy A. Schall has had two patients, Dr. George L. Tobey, Jr., and Dr. Harold G. Tobey one each.

The first is a man 53 years of age who illustrates the difficulty in making a diagnosis. Suddenly while taking a shower he felt dizzy, weak, faint and nauseated, and was in a critical condition with depressed respirations and a fibrillating heart. He was treated for coronary thrombosis in bed for five weeks and then was normal again. Three months later and five months later he had similar attacks. In his fourth attack he developed a nystagmus, which caused him to be sent to an aurist. His vestibular function was impaired in both ears, more marked on the left side, with early perception type deafness in each ear, but more advanced in the left ear, and then Ménière's disease was thought a distinct possibility. He had suffered no tinnitus and had not been conscious of the deafness. He had ten of these major attacks over a 19-month period. Furstenburg's treatment had been tried without any improvement and finally the nerve was sectioned in 1935 and he has been well since.

Another case of a woman, aged 47 years, shows the severe type of Ménière's syndrome but it ran a more typical course. She had a sudden attack of severe vertigo with nausea and vomiting. The right ear was blocked and hearing impaired with unilateral tinnitus. She had recurring attacks for one year. She tried the Furstenburg regime for two months without success and was operated on by Dr. Walter Dandy. Her vertigo was relieved. The tinnitus continues.

Another woman, 55 years of age, had her first series of attacks in 1912. She complained of constant tinnitus with pressure in the right side of her head and occasional dizziness. In 1915 she was again seen with mild, recurring attacks. Apparently there was a remission for twenty-two years, during which time she had a nervous breakdown and a gallbladder operation, among other things. In 1937 the Ménière's attacks recurred, were incapacitating, and persisted for two years in spite of Furstenberg's treatment. In 1939 Dr. Donald Munro sectioned the vestibular portion of her eighth nerve. There was a complete disappearance of the vertigo and a retention of her preoperative hearing.

Spontaneous remissions will occur. They have occurred in 41 per cent of one large series lasting from a few months up to twelve years. Because of these spontaneous remissions it is difficult to evaluate treatment.


128 256 512 1024 2048 2896 4096 5792 8192 11584
Loss in Decibels
BEFORE NERVE SECTION
CENTER SPEECH RANGE
SECONDARY SPEECH TONES
Left
Right
Db. loss Whisp. Voice Air L. R. Bone L. R.

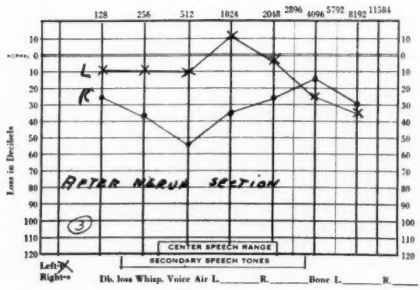

128 256 512 1024 2048 2896 4096 5792 8192 11584
Loss in Decibels
AFTER NERVE SECTION
CENTER SPEECH RANGE
SECONDARY SPEECH TONES
Left
Right
Db. loss Whisp. Voice Air L. R. Bone L. R.

Fig. 3.—Audiogram of hearing before and after operation.

Thornwall observed 15 patients for many years and found that the vertigo lasted for from six months to twelve years and entirely ceased thereafter.

If Ménière's disease were as simple and easy to diagnose and treat and all cases fell into one of two groups as suggested by Miles Atkinson in introducing his method for caloric test, we otologists could contribute more. Atkinson says that Ménière's syndrome can be produced by either of two mechanisms, the one allergic and the other vasospastic. These can be differentiated by means of the histamine skin test, the allergic group giving a positive reaction and the vasospastic a negative one. Here again we hear the echo of water metabolism, for in allergy it is known that the capillary permeability is increased and histamine is known to affect capillary permeability. When the vestibular function came to be correlated with the cutaneous reaction to histamine, it was found that in the allergic group vestibular function was either diminished or unchanged while in the vasospastic group, it was almost invariably increased or hyperactive.

He further shows characteristic audiograms for the two groups. In the allergic group a bilateral conduction type of deafness was noted, while in the vasospastic group perception deafness was invariably seen.

Of 22 patients with the Ménière syndrome on whom a histamine test was done, eight reacted positively and were treated successfully by desensitization.

Shelden and Horton, of the Mayo Clinic, report excellent results in the histamine treatment of Ménière's syndrome. Horton accidentally found, while treating vascular headaches, that histamine relieved a patient with Ménière's syndrome. They give intravenously 1.9 mg. of histamine acid phosphate dissolved in 250 c.c. of normal physiologic salt solution on two or three successive days. The time required to administer the solution is 1½ hours. The 15 patients selected for this treatment had previously received ammonium chloride or potassium nitrate for several months with no benefit. They were also in acute or subacute stages of the disease and were totally or partially incapacitated. One patient had been vomiting continuously for fifteen hours. All were relieved immediately in a spectacular manner. No ill effects were noted, except an occasional slight sensation of heat in the face.

Talbot has tried it in six cases where potassium chloride failed to bring relief, and in these six cases histamine was of no further help.

McNally, of Toronto, finds it unsatisfactory.

The idiopathic Ménière's syndrome is a symptom complex of middle life, occurring most frequently in the fourth and fifth decades. It would seem that oft-repeated, small, explosive vasomotor disturbances in the internal ear tear down and destroy the tonus or rhythm between the two labyrinths, whether by faulty water metabolism or not.

These cases are very often associated with the menopause and with severe emotional upsets and anxiety states where the vasomotor system is deranged.

Potassium and other drugs may readjust the disharmony.

We might compare the tonus of the labyrinths to the rhythm of the heart beat. If either of these rhythms is disturbed, critical clinical symptoms may ensue. Digitalis or electrolyte may be enough to bring back the normal rhythms.

Ballenger and Lewis have made a statistical study of 33 cases diagnosed as Ménière's symptom complex at the Massachusetts Eye and Ear Infirmary. Cases diagnosed as toxic labyrinthitis have not been included.

1. The average age was 48.9 years.

2. The sex incidence was 52 per cent males and 48 per cent females.

3. The average length of time after the development of the first aural symptom until a physician was consulted was 4.6 years.

4. The most common symptom first noticed by the patient was vertigo, which appeared in two-thirds of the cases. Nausea and vomiting, or tinnitus, or deafness occurred as the first symptom in about one-third of the cases.

5. Of the patients 18.2 per cent stated that certain positions of the head either aggravated or precipitated an attack.

6. Bárány and caloric tests showed either unilateral or bilateral hypofunctioning labyrinths in 88 per cent, normal labyrinths in 11.6 per cent, and a nonresponsive labyrinth in one case. There

were no hyperfunctioning labyrinths. Of the hypofunctioning labyrinths 57 per cent were unilateral and 43 per cent bilateral.

7. Audiometric examinations revealed unilateral or bilateral deafness in 100 per cent of the cases, of which 93 per cent had either a nerve or mixed type and only 7 per cent a conductive deafness. Of all cases 90 per cent had a moderate or severe deafness (30 decibels or more).

8. Only two patients had any form of aura preceding the attack.

9. In this series numerous coexisting conditions were found. Arteriosclerosis was present in three; syphilis, infected tonsils and a strong functional element in two each; and sinusitis, chronic cholecystitis, essential hypertension, cystitis and infected teeth in one each.

10. The results of the treatment of these patients were rather inconclusive. Of 10 patients on ammonium chloride and a low salt diet four received no benefit, four had mild and six moderate improvement of symptoms for varying lengths of time up to two years. It should be noted here that these patients were on a low salt diet and not a salt-free diet, as recommended by Furstenberg. In two of these improved patients, attempts to produce an exacerbation of symptoms by elevations of the blood sodium were unsuccessful.

Of 15 patients treated with potassium chloride five received no benefit, six had mild and four moderate improvement of symptoms for a variable length of time up to two years.

Of two patients treated with histamine alone neither were improved, while one patient on histamine and potassium chloride received minimal alleviation of symptoms.

One patient with arteriosclerosis was moderately free of symptoms on phenobarbital alone.

Another patient with a low basal metabolic rate received no relief from thyroid or other forms of treatment.

Removal of foci of infection gave moderate improvement to one patient.

Of two cases coming to surgery for eighth nerve section one patient was not benefited, as difficulty was encountered in locating the eighth nerve, and the other was relieved of vertigo, but the tinnitus persisted.

One patient was moderately relieved by ligation of the ductus endolymphaticus and another was moderately relieved by coagulation of the cochlea and labyrinth.

403 COMMONWEALTH AVE.

BIBLIOGRAPHY

1. Ménière, P.: Mémoires sur des lésions de l'oreille interne donnant lieu á des symptoms de congestion cérébrale apoplectiforme, Gaz. méd. Paris 16:29, 1861. (Cited by Simonton, K. M.: Ménière Symptom Complex, ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY 49:80 (March), 1940.)

2. McNally, W. J.: Symposium on Vertigo, paper read before joint session of the Academy of Ophthalmology and Otolaryngology at Chicago, Ill., October, 1941.

3. Mygind, S. H., and Dederding, D.: Significance of Water Metabolism in General Pathology as Demonstrated by Experiments on the Ear, Acta otolaryng. 17:404, 1932.

4. Furstenberg, A. C.: Ménière's Symptom Complex. Medical Treatment, Tr. Pacific Coast Oto-Ophthal. Soc., pp. 150-160, 1936.

5. Talbott, J. H., and Brown, M. R.: Ménière's Syndrome. Acid-Base Constituents of the Blood: Treatment With Potassium Chloride, J. A. M. A. 114:125-130 (Jan. 13), 1940.

6. Hallpike, C. S.: Some Recent Work on Intralabyrinthine Pressure, Acta otolaryng. 27:229, 1939.

7. Crowe, S. J.: Ménière's Disease. A Study Based on Examination Made Before and After an Intracranial Division of the Vestibular Nerve, Medicine 17:1 (Feb.), 1938.

8. Atkinson, M.: Changes in Vestibular Sensitivity in Ménière's Syndrome and Their Significance, Arch. Otolaryng. 33:969-974 (June), 1941.

9. Shelden, C. H., and Horton, B. T.: Treatment of Ménière's Disease With Histamine Administered Intravenously, Proc. Staff Meet. Mayo Clin. 15:17 (Jan. 10), 1940.

LXIV

AIR PRESSURES IN THE NOSE AND MAXILLARY SINUS UNDER NORMAL CONDITIONS IN DISEASE*†

DELBERT K. JUDD, M.D.

CHICAGO

During the past five years we have witnessed the development of a new and surprisingly effective method of dealing with infections. With the advent of chemotherapy there have been radical changes in both the treatment and prognosis of lower respiratory tract infections, particularly pneumonia, and infections of the middle ear cell system. However, it must be admitted that a corresponding improvement in either prophylaxis or management of upper respiratory infections involving the nose and paranasal sinuses has not been achieved. It is apparent then that further investigation of the problem of paranasal sinus disease is still necessary if we are to accomplish any improvement in its management.

Although there have been many theories advanced to explain the function of the paranasal sinuses, none seem to be entirely satisfactory. For that matter, the value of their existence in the human being could be, and undoubtedly often has been, questioned because of the complications they so often add to upper respiratory infections. But since they do exist and since the adjacent nasal cavities are of primary importance in the development and cure of infections which occur within them, it is possible that further investigation of the relationships between the nasal and sinus cavities may help to establish the principles underlying methods of therapy.

Important contributions to our knowledge of normal physiologic processes and natural mechanisms of defense against infection have been made along three particular lines.

First, the value of ciliary activity and the mucous blanket covering the nasal mucous membrane as a first line of defense against

---

*From the Department of Surgery, Division of Otolaryngology, the University of Chicago.

†Presented before the Chicago Laryngological and Otological Society, Jan. 5, 1942.

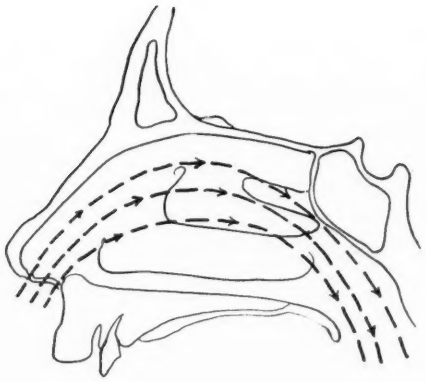


Fig. 1.—Inspiratory air currents are deflected upward by the horizontal opening of the anterior nares to pass in a high curve through the olfactory cleft. This air stream does not enter the meatuses. (Proetz.)

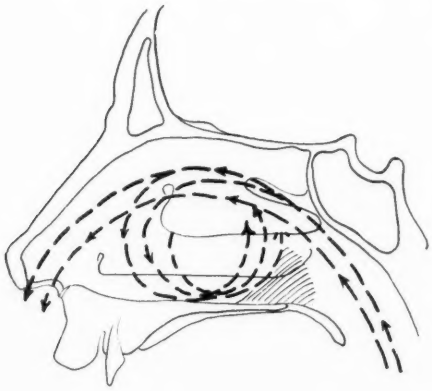


Fig. 2.—Expiratory air currents follow the same high arched curve as inspired air. The outlet (nares), being smaller than the inlet (choana), permits only a part of the air stream to leave the nostril. The remainder is deflected back through the inferior meatus to the choana where the current from the pharynx forces part of it into the middle meatus. (Proetz.)

infection has been amply demonstrated by the investigations of Proetz,[1] Hilding,[2] and others.[3, 4, 5]

Second, the influence of certain qualities of the mucous blanket such as viscosity,[6] hydrogen ion concentration[7, 8] and possible enzyme[9] action upon upper respiratory infection has received considerable investigation.

Third, the existence of still another local line of defense represented in the accumulation of specific antibodies in the subepithelial mucosa has been demonstrated by Walsh and Cannon.[10]

Just as these studies have greatly influenced the development of much of our present-day therapeutic measures, so it is hoped that additional information of practical value will evolve from further study of the more or less fundamental problems such as air currents and pressure changes in the nose and paranasal sinuses under normal conditions and in disease.

In the nose, structure plays a fundamental part in the proper maintenance of physiologic air channels, streams and pressures, limiting excessive ventilation of its own passages and yet directing the air currents in such a way that they are accessible to all portions of the nasal fossa in proper proportions and without too much eddying. In cases of abnormal patency of the nasal passages direct blasts of air may cause drying, halt ciliary activity and thus break down the first line of defense.

Many observers,[11-15] using experiments by means of which the stream of air passing through the nose could be visualized, agree that air currents are determined entirely by the shape of the anterior nares, and that their direction is not altered as they pass over the turbinal structures.

The first two figures are diagrams from Proetz's book[16] and demonstrate readily the mechanism and pathways of air currents through the nasal fossa during both phases of respiration. Inspired air (Fig. 1) instead of passing directly into the pharynx, is deflected upward by the horizontal opening at the anterior nares, to form a high arch as it passes medial to the middle turbinate (outside the middle meatus) and finally being deflected downward through the choana by the front face of the sphenoid. The stream of air completely avoids the lower half of the nasal fossa as well as all meatuses. This explains the nasal stuffiness experienced when the middle turbinate region is blocked, despite the wide air space in the lower half of the nasal fossa. The path of expired air (Fig. 2) follows

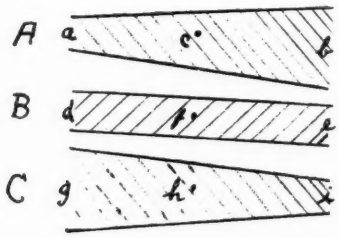


Fig. 3.—Diagrams demonstrating the effects on air pressures at the sinus ostium *(c, f, b)* by variations in the size of the anterior naris *(a, d, g)* or and the posterior choana *(b, e, i)* with resultant alterations in proportions. (After Proetz.)

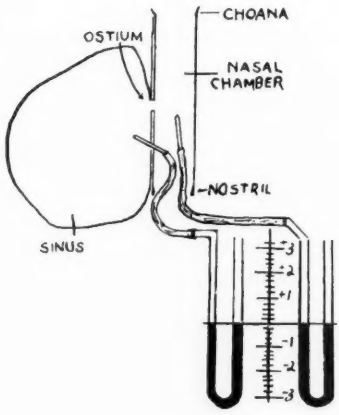


Fig. 4.—Diagram demonstrating apparatus and method used to obtain simultaneous records of air pressures in the nasal cavity and maxillary sinus.

much the same curve but, since the inlet (choana) is larger than the outlet (nostril), the stream of air enters the nasal cavity more rapidly than it can be carried out. This resistance at the anterior nares is sufficient to deflect a portion of the air stream backward through the inferior meatus to the posterior tip of the middle turbinate where it is partially diverted into the middle meatus. Briefly, both inspiratory and expiratory air currents pass in a high curve through the nasal chambers, but only the warm, humidified expiratory currents enter the meatuses. However, it is obvious that the amount of air finally reaching the ostia is so small that very little actual air exchange is possible through the ostia. It is obvious also that the amount of air finally reaching the ostia is so small that very little actual air exchange is possible through the ostia. It is obvious also that such an arrangement makes it impossible for the sinuses to have any part in the warming and moistening of incoming air. The two most important functions of the ostium are: (1) serving as the outlet for the mucous blanket and secretions, and (2) providing a means of air exchange and equalization of air pressure. The performance of these functions is possible only if the ostium is patent and the ciliated epithelium of its mucosa undamaged by drying or cooling. So the protection afforded the ostium as a result of its out-of-the-way location makes possible the existence of a healthy sinus in spite of numerous pathologic conditions which may be present in the nose.

The factors which determine the fluctuations of positive and negative pressures in the nasal cavity (and at the ostia) are illustrated by the diagrams in Fig. 3. With a small nostril or any anterior nasal constriction (Fig. 3*A*) the resistance to the air stream set up by the thoracic bellows is increased. The greater the resistance at the anterior naris *(a)* the greater will be the increase in either the negative or positive pressures created at the ostium *(c)*. As the size of the two orifices becomes more nearly equal (Fig. 3*B*) the intranasal pressures will fluctuate less widely. When the naris *(g)* is normal or large (Fig. 3*C*) and there is some posterior obstruction such as choanal atresia or hypertrophied adenoid, the air stream produced by the thoracic bellows meets resistance at the choana *(i)* and fluctuations in pressure within the nose are reduced to a minimum.

The method and apparatus used in the investigation are illustrated in Fig. 4. The apparatus consists simply of two similar manometer systems, one recording the nasal pressure, the other measuring the intrasinus pressure. Trocars of equal size are used,

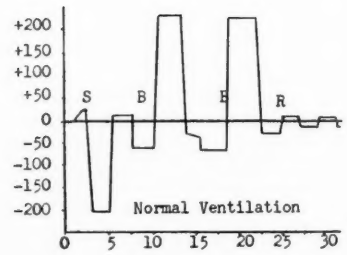


Fig. 5.—When normal ventilation of the nose and patency of the sinus ostium exist there will be simultaneous and equal fluctuations of air pressure in the nose and antrum during any phase of respiratory effort. A single curve represents the pressure changes in both cavities. Vertical line indicates pressure in mm. of water. Time intermal in seconds measured on the horizontal line. *S*—negative pressure produced by sniffing; *B*—positive pressure produced by blowing nose; *R*—pressures during normal respiration.

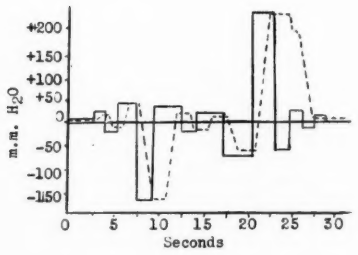


Fig. 6.—Graph showing time interval necessary for air pressure in antrum (dotted line) to equalize changes in pressure within the nasal cavity, when the sinus ostium is partially obstructed.

and since their diameter is not great enough to block the nostril, any interference with the natural mechanism of breathing is minimal. The sinus trocar is passed through the inferior meatus to avoid any interference with the patency of the natural ostium.

This same manometer system was used to measure antrum pressures during the application of mass suction and nasal irrigation. However, whenever either of these was applied to the nostril on the same side as the sinus being tested, it was necessary to use a specially devised rubber nasal tip through which the sinus trocar could also be forced in the direction and at the level required for penetration of the inferior meatal wall.

As previously stated the pressure fluctuations within the sinus cavity are dependent upon the changes in pressure within the nose. There is in addition to this the obvious fact that pressures will equalize on the two sides of the ostium only if this opening is patent; also the ease or rapidity of equalization is determined by the degree of patency.

The various tests made in each case are shown in Tables I, II, III and IV. The figures recorded are the results obtained in a patient with unilateral nasal constriction and partial blocking of the maxillary ostium and represent an average sample of the pressure readings. The observations recorded on Table I serve to illustrate that: (1) With the ostium patent, changes in pressure occuring in the nose, as the result of (a) normal respiration, (b) exaggerated inspiration or sniffing which produces a negative pressure or (c) forced expiration or blowing which produces a positive pressure are transmitted immediately to the sinus cavity. (2) Decreased patency or narrowing of the ostium causes a time lag in the production of pressure changes in the sinus. For example time is required before the sinus pressure undergoes the same change as occurred in the nose. When the ostium is markedly obstructed such extreme pressure changes within the sinus as result from sniffing or blowing will not return to the normal level until forced to do so by the development of exaggerated opposite pressures within the nose. (3) That so long as there is even a small opening through the ostium the peak pressures in the nose will, after an interval, be equalized in the sinus. (4) An observation which will be illustrated later is that when the ostium finally is completely blocked, there will be no pressure fluctuation in the sinus.

Pressure readings were made by carrying out the same tests shown in Table I on a large series of patients which included the

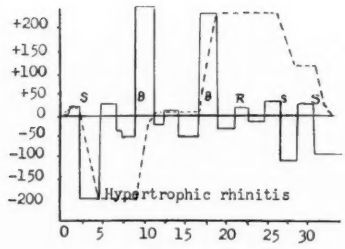


Fig. 7.—Nasal congestion with marked reduction of the patency of the sinus ostium. After considerable time lag the intrasinus pressure equalizes the peak pressure produced in the nose by sniffing *(S)* and blowing *(B)* but it remains at the peak level during normal respiration. Equally extreme opposite intranasal pressure is necessary to reduce the intrasinus pressure to the normal level.

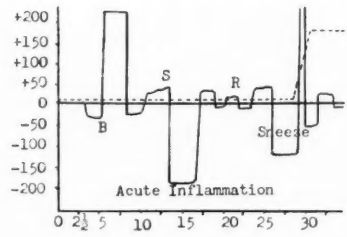
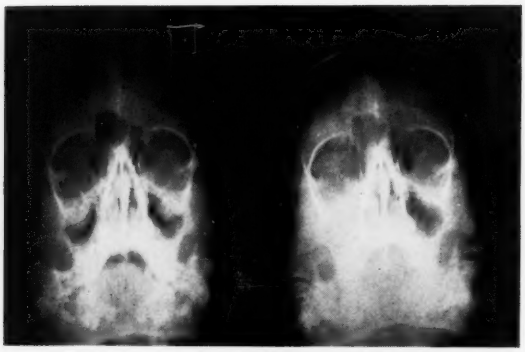


Fig. 8.—Complete obstruction of sinus ostium in acute rhinitis. Antrum pressures (dotted line) remain unaltered despite exaggerated respiratory efforts. Sneezing however created sufficient force to raise the sinus pressure; this increased pressure could not be reduced by sniffing.

following clinical conditions: hypertrophic rhinitis and sinusitis, suppurative sinusitis, atrophic rhinitis, and nasal polypi (before and after removal). The results of these observations are illustrated by a series of curves which show the simultaneous pressure readings in the nasal cavity and maxillary sinus under varying conditions. In the normal nose (Fig. 5) the pressures within both sinus and nasal cavities can be represented by a single curve since the ostium is patent. The pressures are at all times equal regardless of the respiratory effort. With the ostium partially blocked (Fig. 6) a certain length of time is required for the pressure in the sinus to equalize changes in air pressure produced in the nasal cavity. However, the peak pressure in the two cavities does eventually reach the same level. With a further degree of obstruction of the ostium (Fig. 7) the extreme negative pressure produced in the nose by sniffing and the high positive pressure produced by blowing may force the intrasinus pressure to the same level after a time interval, but the narrow ostium does not permit a return to the normal level until exaggerated opposite pressures are developed in the nose.

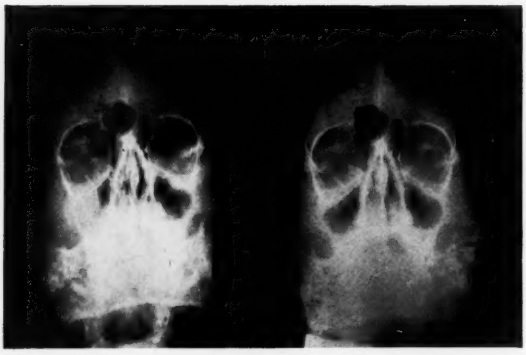
The curves in Fig. 8 were made from readings taken during an acute inflammation which resulted in severe obstruction of the sinus ostium. The exaggerated respiratory efforts producing the marked changes in the nasal pressure did not affect the pressure in the sinus until suddenly, when the patient sneezed, tremendous force raised the sinus pressure to some extent. However, during a fifteen-minute period of observation the change in pressure persisted despite repeated attempts to bring it down by forceful inspiration.

Now during the process of an acute inflammation, the balancing of air pressures is in itself of minor importance functionally, but the observation and study of these pressures makes possible a clearer understanding of the efficiency with which the ostium is performing its major function; namely, the maintenance of an adequate outlet for the sinus secretions which are propelled by the cilia. For example the stages of an acute inflammatory process in the maxillary sinuses as shown in the X-rays (Figs. 9 and 10) will vary in severity depending upon the degree of patency of the ostia. The first X-ray in Fig. 9*A* shows an acute inflammatory process in the nose and maxillary sinuses with the mucosa of both antra uniformly thickened. If the pressure is measured at this time a definite time lag or sustained pressure will be found, indicating the more important fact that there is interference with the flow of secretions through the ostia. One week later the X-ray (Fig. 9*B*) shows that the ostium on one side has been able to maintain and improve its patency so that clear-



A. *B.*

Fig. 9.—The X-ray on the left was taken at the onset of an acute upper respiratory infection and shows the thickened mucous membrane. The second X-ray was taken one week later; note empyema of left antrum.



A. *B.*

Fig. 10.—The first X-ray was taken one week after the second X-ray in Fig. 9; empyema is clearing. The last X-ray one week later; empyema has subsided.

ing takes place; but on the other side the impedence at the ostium has been sufficient to cause retention and as a result, empyema developed. Another week later (Fig. 10*A*) there is evidence of improvement of the empyema. One week after this both antra are clear although the treatment throughout was conservative, i. e., adequate nasal shrinkage. If measured at this time the pressures on the two sides of the ostia would be equal; also changes in pressure would rapidly equalize.

The investigation included observations on: (1) the use of mass suction (negative pressure); (2) simple irrigation (positive pressure); (3) combined suction and irrigation. The observations made under these various conditions are illustrated in Tables II, III and IV, and the figures are a fair representation of a number of actual readings.

While the readings in Table II show that the intrasinus pressure is reduced during mass suction, this is not necessarily accompanied by removal of any fluid content from the sinus. Actual irrigation of the maxillary antrum after either mass suction or combined suction and irrigation had been used, proved by the large amount of pus recovered that very little if any could have been removed by these procedures. Because of the rigid nature of the sinus walls, mass suction will remove fluid from the cavity only if the ostium is patent, air is present in the sinus, and the retained fluid overlies the ostium. In addition the condition of the ostium must be such as to maintain its patency under negative pressures without collapse of its walls. Under these conditions, mass suction can, especially if interrupted, remove part of the fluid contents of a sinus but actual observation shows the method to be of very limited value. It should be pointed out that these observations on the use of mass suction have no bearing on the displacement method of introducing medication into the sinuses.

Table III shows that the positive intranasal pressure produced by simple irrigation is accompanied by an increased pressure in the sinuses. Secretions may be removed from the nostril by nasal irrigation, but not from the sinuses. A possible exception occurs when a tenaceous mass of mucus projects through the ostium into the nasal passage and is subjected to the mechanical force of the irrigating stream. As the exposed portion is moved away by the stream of irrigating solution it pulls with it, as a result of the tenacity of the mucus, that part of the mass still retained in the sinus.

Table IV shows the pressure changes in the maxillary sinus during the application to the nasal cavities of suction and irrigation

combined. The essential difference in this procedure as compared to irrigation with positive pressure, is that the flow of the irrigating solution is maintained by negative pressure created by either a siphon or a pump. The pressures in the sinuses thereby become negative. After treatment by this procedure, including adequate shrinkage at the ostium, the amount of pus recovered from the antrum by irrigation through a cannula demonstrated that little or no discharge had been removed by the combined suction and nasal irrigation.

SUMMARY

1. In the normal nose, pressure equilibrium is maintained between the maxillary sinus and nasal cavity during all phases of quiet or exaggerated expiratory excursions.

2. Nose blowing produces high positive pressure in the antrum and sniffing produces extreme negative pressure.

3. All pathologic conditions which produce swelling of the nasal mucosa or varying degrees of occlusion at the ostium, interfere with maintenance of pressure equilibrium between the nasal and sinus cavities.

4. The interference with transmission of pressure changes through the ostium is an indication of the amount of impedence to sinus drainage by ciliary action.

5. Actual measurements show mass suction to be extremely ineffective in removing secretions from the antrum.

6. Nasal irrigation under positive pressure of fluid removes secretions from the nasal passages, but creates positive pressure in the antrum and does not remove the secretions from it.

7. Suction-irrigation, by actual measurement, does not remove any appreciable quantity of secretion from a maxillary sinus which is filled with pus even after thorough shrinkage of the mucosa at the ostium.

950 E. 59TH ST.

BIBLIOGRAPHY

1. Proetz, Arthur W.: Some Intimate Studies of Nasal Function: Their Bearing on Diagnosis and Treatment, ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY, 41:125-140 (March), 1932; Nasal Ciliated Epithelium With Special Reference to Infection and Treatment, J. Laryng. & Otol. 49:557 (Sept.), 1934; Five Preliminary Notes on Nasal Function, ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY 41:1117-1123 (Dec.), 1932.

2. Hilding, Anderson: Influence of Ciliary Activity on the Bacteriology of the Nose, Proc. Staff Meet. Mayo Clin. 6:285-287 (May 13), 1931; The Physiology of Drainage of Nasal Mucus—The Flow of the Mucus Currents Through the Drainage System of the Nasal Mucosa and Its Relation to Ciliary Activity, Arch. Otolaryng. 15:92 (Jan.), 1932.

3. Gray, J.: Ciliary Movement, New York, 1938, The Macmillan Co.

4. Latta, John S., and Schall, R. F.: The Histology of the Paranasal Sinuses Under Various Conditions, ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY 43:945 (Dec.), 1934.

5. Sternberg, H.: Physiology of the Mucosa (Beitrage jur Physiologie und pathologie der Schleimhaut der Luftwege), Ztschr. f. Hals-Nasen u. Ohrenh. 7:432, 1924.

6. Linton, C. S.: Resistance of the Upper Respiratory Mucosa to Infection, ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY 42:64 (March), 1933.

7. Hilding, Anderson: Note on Some Change in the Hydrogen Ion Concentration of Nasal Mucus, ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY 43:47 (March), 1934.

8. Fabricant, N. D.: Significance of pH of Nasal Secretions in Situ, Arch. Otolaryng. 34:150-163 (July), 1941; Significance of pH of Nasal Secretions in Situ; Further Studies, *ibid.* 34:297-301 (Aug.), 1941; Effect of Silver Preparations and Antiseptics on the pH of Nasal Secretions in Situ, *ibid.* 34:302-306 (Aug.), 1941.

9. Buhrmester, Catherine C., and Wenner, W. F.: Presence of a Histamine-like Substance in Nasal Mucosa, Nasal Polypi and Nasal Secretion, Arch. Otolaryng. 24:570 (Nov.), 1936.

10. Walsh, T. E., and Cannon, Paul R.: Local Formation of Antibody by the Nasal Mucosa, Proc. Soc. Exper. Biol. & Med. 29:675, 1932.

11. Braune, W., and Clasen, F. E.: Die Nebenhohlen der Menschlichen Nase in ihrer Bedeutung fur den Mechanismus des Riechens, Ztschr. f. Anat. u. Entwickelungsgeschichte 2:1, 1876.

12. Hartz, Henry J.: Remarks on the Physiology and Development of the Nose, ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY 18:907 (Dec.), 1909.

13. Paulsen, E.: Experimentelle untersuchungen uber die Stromungen der Luft in der Nasenhohle, Sitzungsb. d. K. Akad. d. Wissenschaft. section III, 1882.

14. Scheideler, J.: The Air Currents in a Human Nose During Respiration, Ztschr. f. Hals,- Nasen- u. Ohrenh. 44:228, 1938.

15. Proetz, Arthur W.: Some Intimate Studies of Nasal Function, ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY 41:125 (March), 1932.

16. Proetz, Arthur W.: Essays on the Applied Physiology of the Nose, St. Louis, 1941, Annals Publishing Co., pp. 116-117.

TABLE I*

SIMULTANEOUS PRESSURES WITHIN NASAL FOSSA AND MAXILLARY SINUS WITH WATER MANOMETER

Conditions	Before Shrinkage				After Shrinkage			
	Right Ostium Patent		Left Ostium Obstr.		Right		Left	
	Nostril	Antrum	Nostril	Antrum	Nostril	Antrum	Nostril	Antrum
Quiet Respiration								
1. *Inspiration*	—4	—4	—6	—6	—4	—4	—5	—4
(Time lag)				2 S.				0
2. *Expiration*	+6	+6	+7	+6	+4	+4	+4	+4
(Time lag)				3 S.		0		0
Exaggerated Respiration								
1. *Inspiration* (sniffing)								
a. R. nostril closed		—15	—180	—180		—10	—150	—150
(Time lag)		0		U.B.		0		4 M.
b. L. nostril closed	—145	—145			—120	—120		—5
(Time lag)		0		0		0		40 S.
c. Both nostrils open	—50	—18	—60	—6	—22	—22	—25	—20
(Time lag)		0		5 M				2 M.
2. *Expiration* (blowing)								
a. R. nostril closed		+290	+250	+250		+250	+200	+200
(Time lag)		0		U.S.		0		8 M.
b. L. nostril closed	+200	+200		+280	+170	+170		+195
(Time lag)		0		U.S.		0		U.S.
c. Both nostrils open	+90	+90	+105	+105	+50	+50	+80	+80
(Time lag)		0		U.S.		0		4 M.

*Form sheet used in recording water manometer pressure readings in each case. Clinical observations of nasal congestion and ventilation of middle meatus, before and after shrinkage, are noted in the column for the nostril involved. Time lag or the interval of time required for intrasinus pressure to equalize intranasal pressure changes is indicated as (S) seconds; (M) minutes (U.B. negative pressure maintained until blowing nose; U.S. positive pressure maintained until sniffing).

TABLE II*

MEASUREMENTS WITH MASS SUCTION. RIGHT OSTIUM PATENT. LEFT OSTIUM PARTIALLY OBSTRUCTED.

Suction Pressure	Applied to:	Pressure R. Antrum	Sust. Pr.	Pressure L. Antrum	Sust. Pr.
152 mm. Hg. or	R. Nostril	—150 mm. Hg.	0	—125 mm. Hg.	U.B.
2,052 mm. H_2O	L. Nostril	—150 mm. Hg.	0	—150 mm. Hg.	U.B.

TABLE III*

MEASUREMENTS DURING IRRIGATION WITHOUT SUCTION

Pressure of of Sol.	Applied to:	Pressure R. Antrum	Sust. Pr.	Pressure L. Antrum	Sust. Pr.
600 mm. H_2O	Right	+10 — +120 mm. H_2O	0	0	0
60 gm./sq.cm.	Left	0	0	+5 to 10 mm. H_2O	about 5 S

TABLE IV*

MEASUREMENTS WITH COMBINED SUCTION AND IRRIGATION

Amount of Suction	Suction Nostril	Irrigation Nostril	R. Antrum	Sust. Pr.	L. Antrum	Sust. Pr.
152 mm. Hg.	Right	Left	—125 mm. Hg.	0	—100 mm. Hg.	0
or 4,052 mm. H_2O	Left	Right	—100 mm. Hg.	U.B.	—80 mm. Hg.	U.B.

*Tables II, III, and IV show how air pressures in the antrum are affected by application of mass suction to the naris (Table II); by the use of nasal irrigation (Table III); and during the use of combined suction and irrigation (Table IV).

LXV

THE TREATMENT OF CONGENITAL ATRESIA OF THE CHOANAE*

V. H. KAZANJIAN, D.M.D., M.D.

BOSTON

Congenital atresia of the choanae is a rare anomaly. Stewart[1] states that 27,863 nasal cases were seen in the Ear, Nose and Throat Department of the Royal Infirmary of Edinburgh during a period of twenty years (1907-1926), and among these there were only six cases of unilateral atresia, and none of his cases were bilateral. The records of the Massachusetts Eye and Ear Infirmary from 1922 to 1938 (in a total of 62,228 cases) yielded 10 cases of atresia which form the basis of this report. Of these 10 cases, four were right-sided, three left-sided, and three were bilateral.

According to Colver[2] the condition was first described by Otto in 1829 following his observations in the dissecting room. The first operation for atresia was performed with good results by Emmert in 1853 on a 7-year-old boy. Since then the literature has contained occasional reports of cases and today the condition and its treatment is fairly familiar to the medical profession.

Developmentally, according to Schaeffer[7] the primitive nasal pouch grows back to meet the ectoderm of the oral fossa. This ruptures through on the thirty-fifth to the thirty-eighth day. Lack of rupture is considered to be the cause of congenital choanal atresia. Bone formation may occur later in this membrane and this may take place even postnatally. With the formation of the face and the palatal process the choanae become slit-like passageways. The growth backward of the palatal processes and septum causes the choanae to move back until they reach the pharynx. The slit-like passageways in this stage between the nose and mouth resemble those found in bony atresia in the adult when it occurs

The condition is not generally considered as hereditary. Colver[2] cites one family in which the mother, two sisters, two daughters and a son were all affected. The group studied at the Eye and Ear In-

*From the Department of Otolaryngology of the Massachusetts Eye and Ear Infirmary.

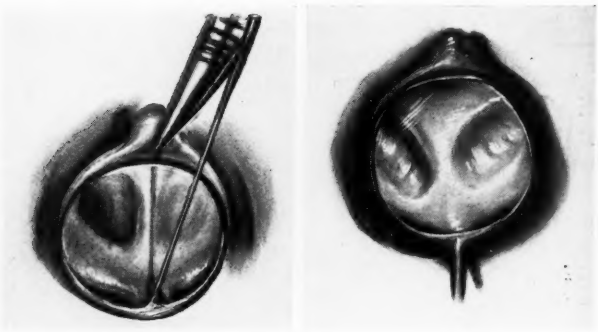
firmary had no familial history of the disease. Facial deformity has been described as accompanying atresia of the posterior choanae, and this condition was present in one of our cases in which there was present a high, wide nasal bridge with an almost bifid tip of the nose. The palatal vault was also very high in this patient (Case 1, Fig. 3).

The classification of congenital atresias is rather simple. They are described as being membranous or bony. But this simplicity in my experience is unjustified since often the obstruction is combined or dual in nature. In our series of cases four had bony atresia, two had membranous atresia, and in four the atresia was bony with a central membranous area.

With this condition there is a fairly definite symptomatology. In the bilateral cases constant mouth breathing is present and irregularities of the teeth and a high palatal vault are usually present. Frequently the nasopharynx shows a dry and atrophic mucosa with an absence of adenoid tissue. In unilateral cases patients having partial functioning of the nose show less marked symptoms than in the bilateral cases. It is perhaps for this reason that we have noted in the history a frequency of tonsillectomies, septal resections and adenoid and sinus operations which had been performed in the past and which suggested that the true condition had escaped diagnosis. Anosmia and frontal headaches are occasional complaints. A constant discharge of mucous secretion is a positive symptom. In infants bilateral occlusion causes great difficulty in breathing and Ronaldson in 1880 reported this to be a frequent cause of asphyxia of the newborn.

The following points are helpful in making a diagnosis of congenital atresia: (1) There is complete obstruction of one or both sides of the nose with no other clear-cut cause such as a badly deviated septum, a foreign body, or tumor. (2) The floor or the nose is covered with a collection of mucous secretions. (3) The passage of a probe into the nasopharynx is found to be impossible. (4) The posterior choanae by inspection or palpation will present a characteristic picture (Fig. 1). (5) An X-ray examination of the nose filled with an opaque oil is diagnostic.

Most operators agree that the best surgical approach for relief of the obstruction is intranasal, beginning with a resection of the septum and continuing further until the obstruction is reached. A few have advocated the removal of the posterior part of the septum. In our series one of the cases was operated upon successfully by Dr.



A. *B.*

Fig. 1.—*A.* Nasopharyngeal "mirror exposure" of a unilateral choanal atresia. *B.* Nasopharyngeal "mirror exposure" of a bilateral choanal atresia.

H. A. Barnes who used an intraoral approach through the hard and soft palates. In my own four cases, three of which had bilateral atresia and one unilateral, the operative procedure is described below.

The incision is made at the base of the columella and the scalpel is carried back along the floor of the nose through the cartilaginous septum parallel to the vomer until the edge of the perpendicular plate of the ethmoid is met. From this point the through-and-through incision is carried almost vertically upward to the roof of the nose. (Fig. 2.) This frees the mobile and cartilaginous septum and it can be held out of the way to one side by a speculum. The bony septum is now removed with its membranous coverings piecemeal, keeping close to the floor of the nose until the obstruction or point of atresia is reached. Usually this obstruction cannot be pierced easily and the chisel must be used to perforate it. Following this the Kerrison forceps is used to remove the obstruction between the nasopharynx and nasal chamber. The cartilaginous septum and columella are now resutured to their original points of attachment and the nose is packed with rubber finger-cot plugs for twenty-four hours.

This procedure yields an excellent exposure of the field so that the entire operation is carried out under direct vision. A single wide posterior chamber is created communicating with the nasopharynx and yielding ample breathing space.

The external columella incision leaves no visible scar if suturing is carried out carefully. No important hemorrhage has been encountered during the operation. Fairly large pulsating vessels are clamped, and to be on the safe side, these may be ligated directly without unusual difficulty.

CASE REPORTS

CASE 1.—J. S., female, aged 15 years. Diagnosis: Bilateral congenital atresia of the choanae—nasal deformity. The patient was admitted to the hospital on April 6, 1936. She primarily applied for treatment of the nasal deformity and routine examination disclosed the existence of bilateral congenital atresia of the choanae in addition to the nasal deformity. This patient is the only one in this series of cases who presented an external as well as internal deformity of the nose. She had a history of nasal obstruction since birth, but did not seem to suffer any great discomfort.

Examination showed the nose to be very wide, especially the parts involving the nasal bones below the frontal eminence and between the eyes. The nasal bones did not appear to come together in the midline, but bulged laterally, leaving a plateau-like dorsum. The tip of the nose was broad with a definite depression in the center giving the appearance of a slightly bifid nose (Fig. 3). There was also a definite defect of the left wing of the nose, with the natural curve missing, probably due to the deficiency or softness of the alar cartilages. The columella was short and the outline of the nostrils round instead of oval. The left naris was partially blocked by a deviation of the septum, and the nasal mucous membrane was pale and edematous with thin serous secretions retained on the floor. No opening could be made out by postnasal examination. The eustachian orifices appeared normal and the larynx also seemed normal. The teeth were irregular and the palatal arch extremely high and narrow.

Operation was performed under ether anesthesia on April 9, 1936. The posterior pharynx was examined and the choanae on both sides were found to be closed, on the left side due to bony stenosis and on the right side bony stenosis was present except for a small opening covered by thin membrane. The operation was first started through the left naris, and the left inferior turbinate was removed; immediately the maxillary sinus was exposed. It was necessary to remove the lateral nasal wall and some of the ethmoid cells. At this stage it was apparent that the approach through the left naris alone would not give sufficient exposure to remove the bony obstruction of the choanae. Therefore, it was necessary to cut through the base of the columella and raise it toward the tip of the nose. Septal resection was carried out as far back as the sphenoid. The abnormality found was such that the floor of the nose on the left side reached half-way to the middle turbinate. The floor on the right side was not as high, but was elevated above normal. Before chiselling the bony obstruction it was necessary to remove a section of mucous membrane flap of the resected septum, anterior to the choana. After obtaining a good exposure of the anterior surface of the bony obstruction the initial opening was made by pointing a chisel close to the floor of the nose and gradually enlarging the opening to sufficient size to allow introduction of a Kerrison forceps to complete the removal of the bony obstruction. A rubber drain was applied.

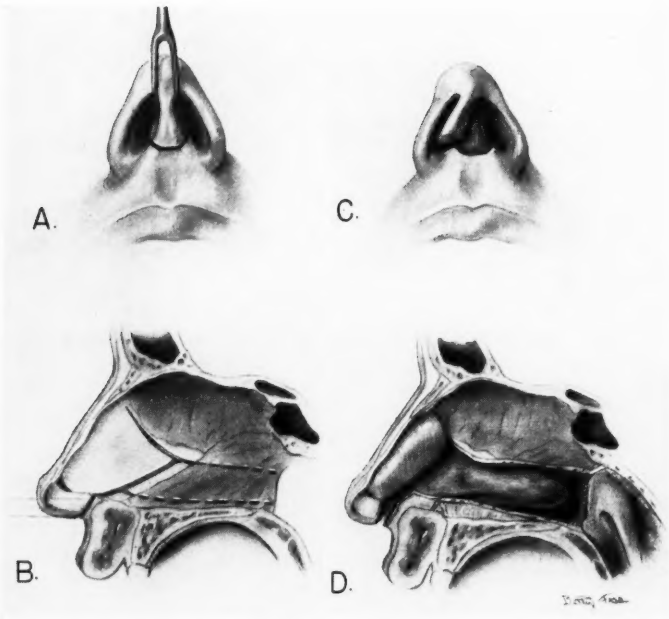


Fig. 2.—*A.* The incision through the base of the columella is in three distinct planes so that when the cut is resutured it will approximate exactly, and leave a minimum amount of scarring. *B.* In extending the through-and-through incision of the septum backward it is carried parallel to the vomer ridge to the tubercle where it turns at right angles to continue through the quadrilateral cartilage up to the nasal bones. *C.* The flap thus created hinges along the dorsum and may be pushed to either side exposing the rest of the septum. It creates a large opening through which an excellent exposure is obtained. *D.* The septum pushed to the opposite side, the vomer removed back to the atresia and then the atresia has been removed laterally to the outer wall of the nose.

The patient had subsequent operations for the correction of the external deformity of the nose. Her last visit to the hospital Out-Patient Department, was on Oct. 9, 1937, one and one-half years after her original visit. Examination at that time showed a permanent opening at the postnasal region and the patient was free from symptoms of nasal obstruction.

CASE 2.—J. E. R., female, aged 9 years. Diagnosis: Atresia of the right choana. This patient was admitted to the hospital Oct. 14, 1937. She had a history of one-sided nasal obstruction since birth. As an infant she had considerable difficulty in breathing which was associated with a good deal of noise. At the age of 3 months an adenoidectomy was done at a local hospital. Following this her breathing was better but there was still some obstruction. The patient had frequent head colds and occasional earache. For the past two years she had had almost constant discharge from the right side of the nose. Headaches had been infrequent. Local examination showed the left nostril to be normal. The right nostril showed presence of purulent discharge and she was unable to breathe on this side. Posterior rhinoscopy revealed no adenoid tissue but a complete occlusion of the right choana.

The patient was operated upon under ether anesthesia Oct. 25, 1937. The face was prepared with metaphen and posterior rhinoscopy was carried out. The right choana was found to be blocked by a bony partition. A postnasal plug was inserted. An incision was made at the base of the columella through the skin and cartilage and carried horizontally and posteriorly along the floor of the septum to within about 1.5 cm. of the sphenoid. The incision was then carried upward to the roof as far as possible in an oblique direction. The septum and columella were then pushed aside, bleeding was controlled and a triangular portion of the septum remaining along the floor posteriorly was removed with biting forceps. The operative procedure was carried to the right and the bony partition was removed to its full extent. The patient was re-examined after removal of the postnasal plug. The columella and skin were sutured in place with kaldermic sutures and boric strips were placed in each nostril. The patient was discharged from the hospital Nov. 1, 1937. Two months later (Jan. 4, 1938) she was seen again and examination showed the choana on the right to be patent and both airways good.

CASE 3.—J. MacG., female, aged 16 years. Diagnosis: Congenital atresia of the left choana. This patient gave a history of a frontal headache for six months with a nasal discharge for years. Nasal obstruction, particularly on the left side had been present for years. She had frequent colds but no history of sore throats. Two intranasal operations for obstruction had been performed without relief, and tonsillectomy had been done at the age of 2 years. On July 8, 1937, a left radical antrum operation had been performed and the findings at operation were normal. On July 15, 1937, cauterization of the left inferior turbinate was carried out, following which the patient was seen again in the Out-Patient Department, and she was still unable to breathe through the left side of the nose. It was found at this time that the patient had an occlusion of the left choana which was both bony and membranous. She was readmitted to the house on July 23, 1937, and operated upon the following day by Dr. Meltzer, who did a resection of the posterior portion of the septum and removed the obstruction of the left choana. Both inferior turbinates were fractured and displaced laterally. The anterior tip of the left inferior turbinate was trimmed with the ring punch. The septum was incised

Fig. 3.—Case 1. Preoperative photographs. This patient presented an external nasal deformity, in addition to bilateral congenital atresia of the posterior choanae.

vertically along the lower portion half-way back and the posterior portion of the vomer removed with biting forceps. A bony partition closing the left choana was removed with the use of biting forceps. The posterior nasal spine was removed with a file. Two months following operation the patient was seen again, and examination showed both choanae open and the airways good. The patient was free from symptoms of nasal obstruction.

It is interesting to note in the above case the fact that the patient had been submitted to repeated operations without discovery of the true cause of the obstruction. In looking over the literature one is impressed by the failure of early diagnosis in cases of obstruction of the choana.

CONCLUSIONS

1. Congenital atresia of the choanae occurs more often than is recognized.

2. Bilateral atresia in the newborn is a very grave condition which requires immediate surgical relief.

3. The operative procedure described gives a good exposure and is especially indicated in cases of bilateral atresia.

475 COMMONWEALTH AVE.

BIBLIOGRAPHY

1. Stewart, J. P.: Congenital Atresia of the Posterior Choanae, Arch. Otolaryng. 13:570-583 (April), 1931.

2. Colver, B. C.: Congenital Choanal Atresia, ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY 46:358 (June), 1937.

3. Stinson, W. D.: Osseous Atresia of the Posterior Choana, Arch. Otolaryng. 15:101-103 (Jan.), 1932.

4. Geiger, C. W., and Roth, J. H.: Osseous Choanal Obstruction, Arch. Otolaryng. 15:441-442 (March), 1932.

5. Asherson, N.: Bilateral Congenital Occlusion of the Posterior Choanae Due to Membranous Diaphragm, J. Laryng. & Otolaryng. 45:344-346 (May), 1930.

6. Richardson, C. W.: Congenital Occlusion of the Post-Nasal Orifices With Report of a Case, Tr. Am. Laryng. A. 35:226-231, 1913.

7. Schaeffer, J. P.: Atresia of the Choanae, Laryngoscope 43:480-481 (June), 1933.

8. Schaeffer, J. P.: The Nose, Paranasal Sinuses, Nasolacrimal Passageways, and The Olfactory Organ in Man, Philadelphia, P. Blakiston & Sons.

LXVI

THE DEGENERATION AND ABSORPTION OF THE ORGAN OF CORTI IN ANIMALS*

M. H. Lurie, M.D.

Boston

It is an accepted fact that the organ of Corti degenerates and is absorbed as a result of traumatic damage, but the mechanism of this process is not understood. A large number of animals were studied in an endeavor to obtain evidence of this process.

These animals, chiefly guinea pigs, may be divided into the following groups: (1) animals that have had a surgical operation performed on the cochlea; (2) animals that have been exposed to a tone of sufficient intensity to damage the organ of Corti or throw it off the basilar membrane; (3) animals that have inherited deafness.

In animals that have had surgical operations on the cochlea, the degeneration of the organ of Corti at the region of the lesion is coincident with an ingrowth of fibrous tissue into the scalae media, tympani and vestibuli. With the ingrowth of fibrous tissue, new blood vessels grow in and new formations of tissue take place. In other parts of the cochlea the organ of Corti degenerates and may be absorbed completely without any fibrosis present or any infiltration of endothelial or phagocytic cells. In fact, the cells of the organ of Corti appear to melt away. Fig. 2 shows the end result in a cochlear turn that had a surgical operation.

Animals that were exposed to the traumatic effects of an intense tone also showed degeneration and absorption of the organ of Corti. In a number of these experiments the organ of Corti was thrown off the basilar membrane and was found floating in the scala media. The organ of Corti also showed the traumatic effects for some distance on either side of the lesion produced. Animals were examined a number of days after exposure to an intense tone and certain interesting facts were noted in the study of these animals. There was no evidence of any invasion by fibrous tissue or endothelial cells of any of the scalae of the cochlea. There were no phagocytic

*From the Department of Otolaryngology of the Harvard Medical School and Mosher Laboratory of the Massachusetts Eye and Ear Infirmary.

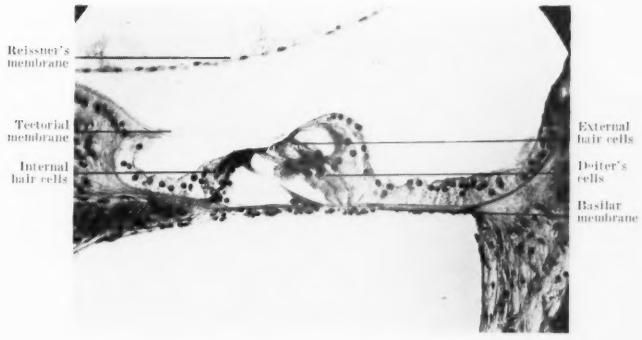


Fig. 1.—Normal organ of Corti of guinea pig (x180).

cells in the scala media in the region where the lesion was produced. The organ of Corti, which had been thrown off from the basilar membrane, had disappeared and no evidence or trace of it could be found in the scala media. The organ of Corti was degenerating and being absorbed in various stages in either direction from the lesion produced. The degeneration of the organ of Corti appears to be a disintegration of the hair cells and their supporting structures. The reticulum of the hair cells and of Deiters' cells seems to have disappeared or to have become broken up. The result is an organ of Corti with a more or less normal outline but with cells that have become completely disorganized. These same regions examined when longer periods of time had elapsed after their exposure showed complete absorption of the organ of Corti often with only a single layer of cells covering the basilar membrane (Figs. 3, 4 and 5).

Trypan blue was injected into a number of these guinea pigs in an endeavor to ascertain if there were any endothelial cells present in the degenerating and absorbing organ of Corti. In all guinea pigs with trypan blue injection studied from one to two weeks after exposure, no evidence could be found of endothelial cells present in the scala media. A large number of endothelial cells filled with trypan blue granules were found in the outer periosteum of the cochlea and in the region of the eustachian tube, but none were found in the scala media, scala tympani or scala vestibuli. No endothelial cells were found in the stria vascularis. Up to the present

Fig. 2.—End result of an operative lesion on the cochlea, resulting in a marked proliferation of connective tissue filling scala tympani and scala media, and complete degeneration of the organ of Corti. This fibrosis extended up toward the helicotrema.

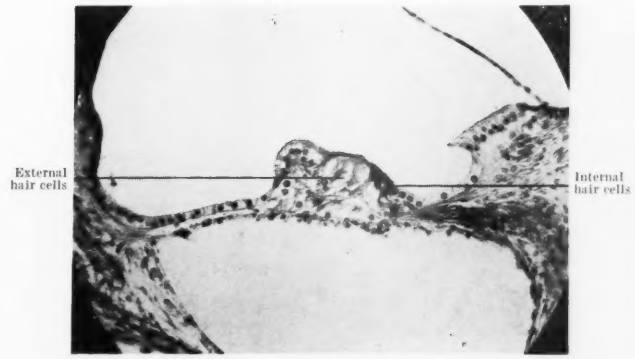


Fig. 3.—Organ of Corti of guinea pig (x150). Seven days after a traumatic exposure to a tone. This section is not at the site of the actual lesion caused by the tone but a half turn above the lesion. Note the complete disintegration of the hair cells and their supporting cells.

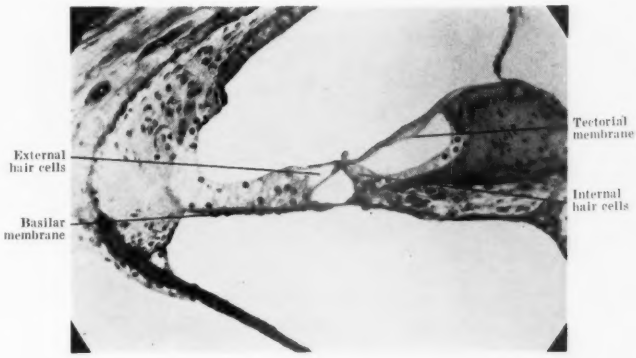


Fig. 4.—Organ of Corti of waltzing mouse (x150). Degeneration and absorption of external and internal hair cells. Degeneration of cochlear nerve. No evidence of a fibrosis or invasion of phagocytic cells.

Fig. 5.—Cochlea of guinea pig 33 days after exposure to a traumatic tone. Showing complete degeneration and absorption of the organ of Corti, degeneration of cochlear nerve and partial degeneration of the spiral ganglion. Note no evidence of any fibrosis or infiltration of cells in the scala media.

time there has been no evidence that the organ of Corti is absorbed by any phagocytic cells or by an invasion of endothelial cells.

The degeneration that takes place in these experimental animals is of the same type that occurs in animals which inherit deafness. This process has already been described in an article, "The Waltzing Guinea Pig."[1] Waltzing guinea pigs injected with trypan blue also gave no evidence of endothelial cell invasion of the scala media or of the organ of Corti. The waltzing (dancing) mice show the same changes as the waltzing guinea pig, that is, a degeneration and absorption of the neural sensory epithelial cells first and then a degeneration of their supporting structure (Fig. 4).

The tectorial membrane in all the animals studied was not involved in this process and appeared to be unaffected by the changes occurring in the scala media. The stria vascularis is usually normal or if there are changes, they do not give any clue as to what occurs in the organ of Corti. The cochlear nerve from the organ of Corti to the spiral ganglion does not begin to degenerate until two or three weeks after the degeneration of the sensory hair cells (Fig. 5).

At the present time the only conclusion that can be drawn from these studies is that a molecular disintegration of the cells of the

organ of Corti takes place. The chemical constituents of the cells dissolve into the fluid of the scala media. This raises the interesting question as to the possible toxic effect, the resulting chemical changes in the scala media may have on the sensory hair cells that were not originally damaged. These toxic chemical changes in the scala media may be the explanation for the continuing and spreading of the degeneration of the organ of Corti throughout the cochlea. This degeneration progresses for long periods of time after the actual injury has occurred.

483 Beacon St.

REFERENCE

1. Lurie, M. H.: The Waltzing (Circling) Guinea Pig, Annals of Otology, Rhinology and Laryngology 50:113 (March), 1941.

LXVII

ROENTGEN FINDINGS IN ACUTE INFECTIONS OF THE MASTOID CELLS*

A. S. Macmillan, M.D.

Boston

With the advent of chemotherapy in the treatment of acute otitis media, the importance of repeated x-ray examinations of the mastoid cannot be overemphasized, as this method of examination may be the only one that can be relied upon, especially if chemotherapy has been administered after the mastoid cells have become involved.

While the findings differ from year to year, with the variations of the infecting organisms, in general they may be classified as those produced (1) when the infection is limited to the mucous membrane lining of the cells and (2) when infection has been present long enough to show bone change. Before considering the changes in the petrosal cells, let us review briefly the changes to be looked for in the mastoid process with relation to infection. Within the first few hours of an acute otitis media, a definite change can be noted in the cells about the antrum. This increased density is the result of absorption of air and an outpouring of serum which gradually spreads and involves all of the cells in the mastoid process and the petrous pyramid. During the first ten days, the density of the mastoid cell area becomes more marked day by day, as the inflammatory edema of the bone and the subperiosteal thickening increase; this characterizes acute inflammation within a cavity lined with mucous membrane whether that cavity is one of the accessory sinuses or one of the mastoid cells.

From the tenth to the fourteenth days, when the otologist is most desirous of knowing the progress of the infection, the patient is usually sent to the roentgenologist for an opinion. Decalcification of the cell walls takes place at this time, due to the increased blood supply to the area, the result of infection. It is now that very care-

---

*From the Department of Otolaryngology of the Massachusetts Eye and Ear Infirmary.

ful attention to technic is essential. Great care should be exercised to duplicate the milliampere seconds and the kilovoltage of the normal and diseased side; furthermore the two radiographs should be made on the same film. Otherwise, the very slight changes due to infection may be overlooked due to slight variations in the screen speed and dark-room technic.

There is no other part of the body where so much depends on comparison of two corresponding areas; there is no other place in the body where such important changes are taking place in so limited an area.

If a radiograph has been made earlier, the radiologist, unless he has been careful in his examination, might have been misled by the lessened density of the cells in question, and might have considered this a clearing-up process rather than a progression of the infection; i.e., the total density is diminished because of the decalcification of the bone.

As a rule, if chemotherapy is instituted after the mastoid has become infected, the clinical symptoms have the appearance of regression, whereas the radiograph shows a condition of progression.

The decalcifying process may extend over a period of a week before coalescence of the cell outline is noted. Most of the early change takes place about the antrum; later on those of the mastoid process and the petrous pyramid become involved. For this reason it is desirable to have more than one projection of the cells in question; i.e., it is as important to have an anteroposterior view of the cell area in addition to the lateral view or Law's position, as it is to have an anteroposterior and a lateral position of a joint or a posteroanterior and a lateral view of the chest.

There are many temporal bones whose cellular development never extends into the mastoid process and it is necessary to take a routine position to show the periantral cells. For years, it has been my practice to take the fronto-occipital projection routinely (Fig. 1). This shows the periantral region and also allows a comparison of the right and left sides of the same exposure.

In the Law position (Fig. 2), the bony capsule and cortex completely obscure the periantral cells. Many times the lateral projection leaves one with the impression that the mastoid is sclerosed, whereas the limited cellular distribution can be readily appreciated in the fronto-occipital projection.

Fig. 1.—Fronto-occipital projection. On the right may be seen the periantral cells—few in number with no history of infection. In the same region on the left is a group of completely broken-down cells, the result of three weeks' infection. In the lateral projection both mastoids appeared to be sclerosed.

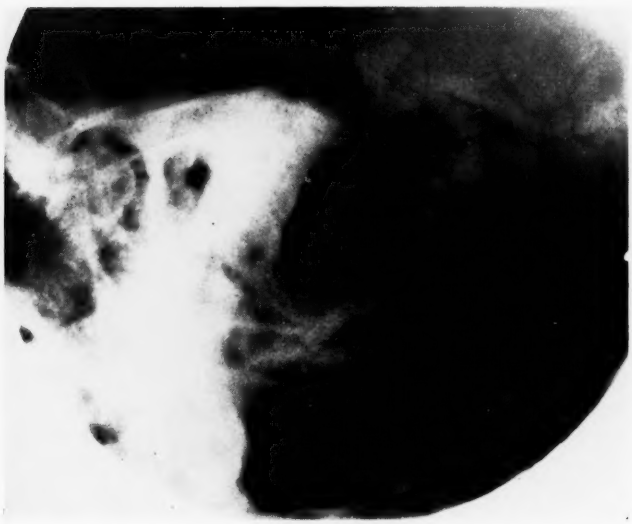


Fig. 2.—Lateral projection of left mastoid shown in Fig. 1. The bony capsule and heavy cortex completely hide the broken-down periantral cells.

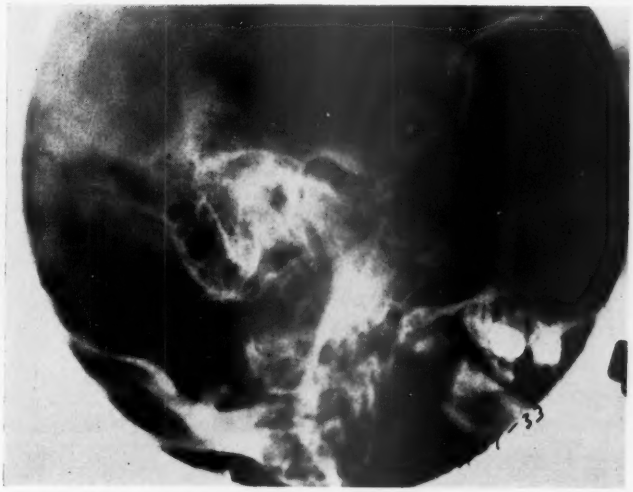


Fig. 3.—Mastoid cell area in a patient with an otitis media of eight days' duration. It can be seen that while the mastoid is definitely infected, there is no decalcification.

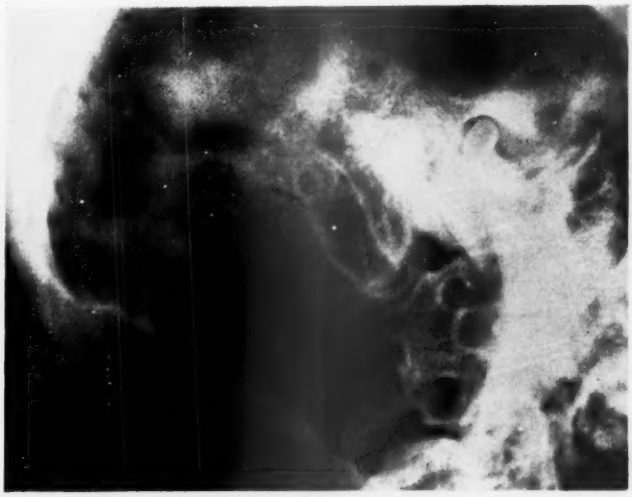


Fig. 4.—Same patient as shown in Fig. 3. Seven weeks after onset, the cell area is completely broken down. No clinical symptoms. No operation. This patient did not have chemotherapy.

Fig. 5.—Same patient as shown in Figs. 3 and 4, nine months after onset The broken-down area has now completely filled in with new bone.

A certain number of patients present themselves at the end of a month or more after the onset of the acute otitis media with the symptoms entirely abated except for the loss of hearing. At this time the roentgen examination may show a complete destruction of the cells. If the patient has been under chemotherapy, operation invariably reveals the area completely filled with pus and granulation tissue. If the patient has not been under chemotherapy and there are no clinical symptoms, the chances are that this broken-down area is sterile. In either case this is an extremely dangerous condition, because, with a new infection, important structures are widely exposed and rapidly developing complications may result. However, nature can take care of this condition within a remarkably short period of time with complete sclerosis in a few months, as shown in Figs. 4 and 5.

Fig. 3 shows the condition of a definitely infected mastoid at the end of eight days. Fig. 4 shows the condition of the mastoid seven weeks after onset, in which case no chemotherapy was instituted; there was a complete clearing up of all symptoms except a resultant loss of hearing. Fig. 5 shows the same patient nine months after onset with a completely sclerosed mastoid.

Fig. 6.—Fronto-occipital projection showing decalcification in the superior medial cells.

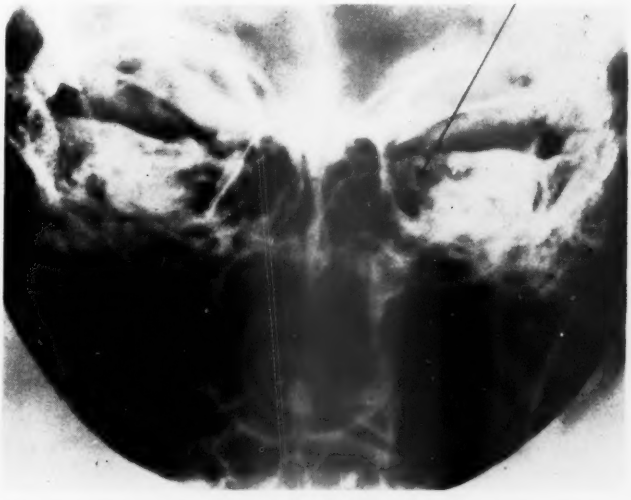


Fig. 7.—Transorbital view of the petrous apices. Decalcification on the right, the infection having reached the apex through the peritubal cells, the only group developed in this patient.

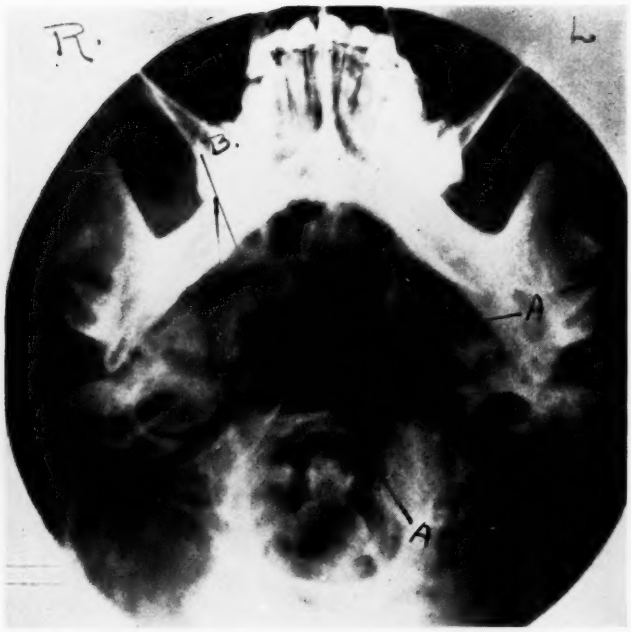


Fig. 8.—Vertex mental projection with very complete destruction of the peritubal cells *(B)* and showing the characteristic swelling of the soft tissues in the nasopharynx *(A)* so often seen when this group of cells are infected.

When infection of the petrous cells is suspected, either because of clinical symptoms or a previous knowledge of extensive pneumatization of the petrous pyramid, it is necessary to take several positions to show the various parts of the pyramid to the best advantage.

As most infections of the petrous pyramid involve those cells extending along the superior surface of the pyramid or its posterior surface, the fronto-occipital projection, taken as a routine procedure, is perhaps the one position of greatest value. Remembering that infection travels in this area, as well as elsewhere in the skull, by thrombophlebitis and continuity of cell structures, it is possible to have an extensive destructive lesion in the cells beyond the bony capsule before there is any destruction of the cells in the mastoid process.

The process of increased density of the petrous cells closely parallels that of the cells in the mastoid process. Decalcification, however, takes place at a slower rate, and if a simple mastoidectomy is performed in a person with an extensive pneumatization of the petrous pyramid, decalcification of the cells of the labyrinth may take place. If the operation is delayed, three separate projections of the petrous pyramid must be made in order to show up the different parts of the pyramid to the best advantage.

The first of these positions is the one already mentioned; i.e., the fronto-occipital projection (Fig. 6), which shows to advantage the cells bordering on the posterior fossa. In temporal bones, where these cells are developed, increased density and decalcification can be as readily determined, as can the cells in a routine mastoid projection.

The majority of cases of petrositis that I have seen show involvement in this area; hence I have made the fronto-occipital projection a routine procedure in the x-ray examination of all mastoids. This position is used in chronic as well as acute mastoiditis, because it shows early enlargement of the antrum due to cholesteatoma formation in chronic otitis media.

The superior group of cells or those beneath the superior surface of the petrous pyramid are best shown in the anteroposterior projection.

In the second position (Fig. 7) the petrous pyramid is shown through the orbit. This also gives an excellent view of the extreme apex and shows decalcification when the infection has reached the apex by either the peritubal, posterior or superior cells.

The third position (Fig. 8), the vertex mental position, shows the pyramid as part of the base of the skull. This shows to advantage the development of the cells by way of the middle ear and eustachian tube. It also shows increase in the soft tissue swelling of the nasopharynx which so frequently accompanies infection of the inferior group of cells.

All these three positions allow a favorable comparison of the infected and the normal side, on the same exposure. Other positions advocated, such as the Stenvers or Yoshies position, in which there is a change in the position of the patient's head to get the two sides on the same plate, makes exact duplication difficult. One cannot limit oneself to one position in the examination of the petrous pyramid as these three positions show different groups of cells.

If swelling of the soft tissue of the nasopharynx cannot be appreciated in the vertex mental projection, a lateral view of the nasopharynx may show it.

While the roentgen examination of the temporal bone is only one link in the chain of evidence the otologist takes into consideration in his examination, it is an important one; every patient with clinical signs of petrositis will show radiographic symptoms if the radiographs are taken with proper attention to positioning and exposure.

483 Beacon St.

LXVIII

HEARING IN CHRONIC OTITIS MEDIA*

PHILIP E. MELTZER, M.D.

BOSTON

Many contributors to this volume dedicated to Dr. Harris P. Mosher have lived their entire otolaryngological existence under his guidance at the Massachusetts General Hospital and the Massachusetts Eye and Ear Infirmary. As one so brought up, I would like to relate one of the developments in the field of otology during his period as Chief of Staff.

My subject deals with the preservation of hearing in chronic otitis media by surgical treatment. This subject was selected because it was my good fortune to have served under such pioneers in middle ear surgery at this hospital as Dr. Frederick L. Jack, Dr. D. Harold Walker and Dr. Harris P. Mosher.

The past twenty-five years have seen many surgical advances in the treatment of this condition. There is still, however, no operative panacea for chronic suppurative otitis media that will eradicate the disease and preserve the hearing or what remains of it.

There will be many shortcomings in a narrative of this kind but as this paper is a report of an experience and not a treatise, details for the most part will be omitted.

What general factors should be kept in mind in offering a prognosis to the patient in regard to hearing? One must refer to the observations known to the older otologists who were so well grounded in the pathology of the middle ear. These men have demonstrated what happens to the mucous membrane in and about the oval and round windows in cases of chronic suppurative otitis media. Crowe,[1] Fowler,[2] and others, have restated these observations in relation to hearing. They have emphasized that a patient with inner ear involvement cannot expect to have his hearing improved by any kind of surgery. Improvement of hearing cannot be expected where fibrous tissue has fixed the stapes in the oval window, or where the round window is impeded by dense, well-epithelized tissue.

---

*From the Department of Otolaryngology of the Massachusetts Eye and Ear Infirmary.

At this point, let me explain what I mean when I speak of hearing in the case of the chronic ear. Is it excellent, good, fair, poor or very poor? Excellent hearing for the purposes of this discussion means to me a threshold of 10 to 15 decibels of loss from the normal in the important frequencies. Stated in another way, it is the degree of hearing which permits a patient to carry on in an absolutely normal way as far as the affected ear is concerned. Good hearing means the patient has a loss of 20 to 30 decibels wherein he hears moderately low conversation from eight to ten feet or slightly beyond that. Fair hearing means that the loss is around 30 to 35 decibels. Conversation can be maintained intelligently at a distance of five to six feet. Poor hearing would be indicated by a loss of 40 to 50 decibels, conversation heard by focusing attention at about two to three feet. Very poor hearing would indicate a loss of over 50 decibels where the patient hears only the raised voice within a few feet and loud conversation four to five feet at best.

It is surprising to note on occasion how good the hearing may be where the malleus and incus appear to be fused by chronic inflammation. It is also remarkable to note occasionally how long the discharge may have continued—years and years in fact—without great interference with the hearing acuity, in cases where the disease has been limited to the attic. Many patients with good or fair hearing may lose it after operation. This is particularly true if during the operation the pathology is such that it is necessary to remove diseased membrane from the regions of the round and oval windows. Scarring in these vital areas, in my opinion, depresses the hearing from its previous level. Many patients could reasonably be assured of retaining what hearing they have if it could be determined from the examination that surgical trauma would be unnecessary in this area. You cannot, however, assure the patient of a dry middle ear.

At this point, I should like to say a word concerning the variations in the character of the discharge and their significance. I, for one, do not hesitate to assure a patient of the harmlessness of a slight mucoid discharge which may at times be considerable in amount, particularly after a nasopharyngeal infection. This type of discharge may even have a slight odor which promptly clears up with a few treatments. In young children with chronic otitis media, where radical operation would be necessary to clear up the discharge, I would much rather preserve the hearing with the eradication of the disease focus by simpler means, than to jeopardize the hearing by a radical in an effort to obtain a dry middle ear.

Lillie[5] has frequently stressed the importance of obtaining a good history and states it is of equal importance with the clinical examination as a factor in determining the surgical procedure. Some of the important inquiries are:

What was the cause of the disease as far as it could be determined? Did it follow one of the diseases of childhood, like scarlet fever, measles, etc? Infections of this kind may be chronic from the very onset. What was the duration of the discharge? Was it months, or years? The nature of the discharge is important. Is it purulent, mucopurulent, thin, scanty, seropurulent or sanguinous? Is the discharge constant or intermittent? What was the longest interval during which the ear was ever dry, and what precipitated the recurrence of the discharge? Is there an odor? Does the odor disappear and the discharge lessen considerably with one or two medical treatments? Does the hearing fluctuate or it is about the same at all times? Under what conditions does it become worse?

Questions pertaining to possible complications are next in order. Has there ever been any dizziness? What is the exact nature of the dizziness? Was it transitory? Did it occur when the ear was being wiped out? Did the dizziness persist for a period of moments, hours, or days? Did the patient have to go to bed or give up work because of the dizziness? Can the patient make sudden motions?

Inquiry as to acute exacerbations is important. Be sure the patient understands what you mean by an acute exacerbation of a chronic ear. Did the ear pain like an acute abscess followed by a profuse discharge? Was it accompanied by tenderness and pain behind the ear? Did the patient have a rise in temperature? How many such attacks had he experienced? Were there ever any chills or chilly sensations during these attacks?

Does the patient suffer from headache? What is its nature? Is it localized in the side of the affected ear? Is it sickeningly severe associated with nausea or with vomiting? Or is the headache so transient or trivial that an aspirin relieves it and the patient is no longer concerned with it? Is the headache present when the ear is free from discharge?

My readers will immediately recognize the importance of obtaining a good history. Who would dare be conservative in the face of symptoms indicating the strong possibility of intracranial complications? To act conservatively in such cases would be like playing with a time-fused bomb which for the moment looks like a dud.

The examination should be carefully made. Minute observations of the appearance of the membrana tympani and the nature of the discharge are of equal importance with the history.

Audiometer tests supplemented with fork tests are desirable. For practical hearing, as far as conversation is concerned, the voice test is utilized. If the McFarland records and the equipment to use them are available, they are to be preferred.

Careful study of the X-ray films in the various positions should be made. Where the X-ray evidence strongly points to a cholesteatoma cavity and the hearing is only fair, it is best to think twice about conservative methods for the preservation of this hearing. On the other hand, with X-ray evidence of cholesteatoma in an ear with good hearing and in the absence of worrysome symptoms, greatest consideration for the preservation of the hearing should be given. I no longer consider the mere presence of cholesteatoma in such cases as an absolute indication for the complete radical operation.

The examination of the fundi and the fields of vision is a routine procedure, particularly if headache or dizziness has been elicited in the history.

Even though basic principles are generally well established they are not inflexible. Most writers on the subject and most surgeons with a large clinical experience agree with the principle that symptoms suggestive of complications as a result of chronic suppurative otitis media should be treated by a complete radical operation.

It is generally agreed that in uncomplicated chronic otitis media with evidence of cholesteatoma, where the hearing is not significant, the complete radical mastoid should be recommended. In an uncomplicated case of chronic suppurative otitis media where the disease is localized to the middle ear, epitympanum, or antrum, and where the hearing is poor, a complete radical is advisable. In uncomplicated chronic otitis media, where the hearing is fair, good or even excellent, the retention of the hearing is of paramount importance. This is the ideal case for some kind of modified radical operation.

The modified radical operation has been repeatedly performed in patients with good hearing, where the disease has been limited to the attic or antrum region, even in cases of circumscribed labyrinthitis with fistula.

Where there has been considerable loss of drum membrane, however, with evidence of ossicular disease, and hearing is fairly

good, a modified radical is contraindicated. Ossiculectomy or regular radical is the procedure of choice.

After careful study of the case it is desirable to begin with a fixed plan of procedure, particularly if you are performing a modified operation. This type of operation requires a step-by-step advance which should not be deviated from if one is to obtain the best results. If, after entering the area the findings warrant a change of plan, it is very simple to do the complete radical operation.

Dr. Mosher very often would say in the discussion of ossiculectomy that "any case of chronic suppurative otitis media that was operated on by ossiculectomy would do better with a radical." This is a very excellent general statement, as it forces one to be ultracautious in the selection of a case for ossiculectomy.

At one time ossiculectomy was one of my favorite procedures and I have been quite satisfied with it. Even though Dr. George L. Tobey has extended this operation into what might be called a middle ear radical, its scope is limited. Ossiculectomy is useful in cases of chronic otitis media with large perforation, where the discharge is apparently due to disease of what remains of the ossicles or of the outer wall of the epitympanum. If there is any evidence by X-ray of enlargement of the antrum it should not be done. It should not be performed where the external canal is small and does not permit full visualization for the necessary instrumentation. It should not be performed where the mucous membrane of the middle ear is badly diseased as indicated by recurring granulations and polyps.

Specially designed middle ear instruments intended for this work are essential. The operation can be performed under local anesthesia by using novocaine 2 per cent solution E Metz. A long, very fine needle, offset at an angle, is used, so that the point can be seen during the injection. The largest sized speculum that completely fills the canal should be used to give maximum visualization. One minim, two at the most, injected in the superior canal wall will anesthetize and blanch this area. A single drop along the floor assures complete anesthesia. A paste of cocaine crystals with adrenalin is applied with an applicator to the mucosa of the middle ear.

With a sharp paracentesis knife, what remains of the drum is severed close to the annulus. Assuming that the right ear is being operated upon, an incision is made in the superior canal wall about

1 to 1½ mm. from the margin of the annulus in a sweep from about nine o'clock to a point at about two o'clock. The membrane in front of the incision is now pushed toward the middle ear and is removed cleanly without tugging, leaving a strip of bare bone corresponding to the outer wall of the epitympanum.

A right angle tenotome is inserted behind what remains of the malleus and the tendon of the tensor tympani is severed. With a special forceps the neck of the malleus is grasped and the ossicle is removed. The incus is now sought with an incus hook and removed if present. Often the incus has sloughed out in the course of the disease.

Now with a long slender, but strong, middle ear Citelli punch or any other fine backward biting forceps the bone filling in the notch of Rivinus is removed. With each bite of the bone in this area the epitympanic space begins to open up into a freely accessible area. Enough bone should be removed so that the anterior end of the lateral semicircular canal and the antrum area can be seen. The cavity is irrigated with dilute peroxide of hydrogen solution, thoroughly dried, and then dusted with a powder blower containing equal parts of sulfanilamide and sulfadiazine. A sterile wick, coated with a light covering of ointment, is loosely placed in the canal. The subsequent dressings are for the purpose of softening the clot that forms in the canal. Warm dilute peroxide solution is used; the canal is dried, and again dusted with the same powder. This procedure in a well-selected case should result in a dry ear with the hearing unchanged in a matter of a few weeks.

Before entering into a discussion of the radical mastoid or of any modification of it as a means of preserving the hearing in the chronic ear, I would like to express my views concerning the postauricular incision versus the Lempert endaural incision for approach to the mastoid and middle ear regions. To decide this point, one must have an experience with both. This experience should not be obtained in a haphazard manner. An apprenticeship is necessary under someone who has seen and understands the whys and wherefores of these incisions and to a degree knows the pitfalls when they are improperly performed.

It has been stated to me by such men as are known to be masters of the radical and modified radical operations by the postauricular route, that they have found the endaural approach highly satisfactory and advantageous.

If you are satisfied with what you accomplish by the postaural method and any other approach holds no interest for you, by all means remain with it. I speak for myself when I say that it would be impossible for me to accomplish what I now am able to do by any other method than the endaural approach, particularly for the modifications of the radical. I reserve the postaural incision in the surgical treatment of chronic otitis media for such conditions where brain abscess is suspected and sought, where the lateral sinus is involved, or in diffuse meningitis. I find the endaural approach most satisfactory in the surgical treatment of chronic otitis media with complications such as labyrinthitis, petrositis or facial paralysis.

There have been a number of modifications of the endaural incision. Kettel,[4] Williams[5] and Shambaugh[6] have described them. It was evident that the skin of the external canal could be utilized as a graft in the radical mastoid operation. Lempert, in his original discussion, sacrificed a good deal of this skin for a direct approach to the antrum. By preserving the skin of the canal in using the endaural approach, an almost complete covering over the facial ridge and part of the mastoid bowl is obtained. This prevents troublesome bridging by granulations from the tegmen to the ridge. The radical operation itself is performed in whatever manner one favors.

There is no question but what experienced technicians can do a great deal in the chronic ear where the disease is limited to the antrum and epitympanum by means of extensive simple mastoidectomy.

This has been discussed by Smith[7] and should be given a trial. If, however, recurrent acute otitis media results in a flare-up of the postaural tissues, I would then convert this operation into a modified radical, which is a very simple thing to do endaurally. Personally I have limited myself in these cases to exteriorization of the epitympanic space without Smith's preliminary trial. In other words, I perform the modified radical immediately. There are definite advantages, however, to what Smith suggests. I have not had any experience with his method of approaching this problem, therefore I cannot take a stand against it.

Cases of chronic otitis media with fair or good hearing, in which the disease is limited to the epitympanic space, are ideally suited for the modified operation. In these cases the perforation is in Shrapnell's membrane; the rest of the membrana tympani appears quite normal or at the most simply thickened with loss of luster, there being no indication that the tympanum proper is affected.

If you are using the endaural approach to expose the antrum and epitympanum, care must be taken not to traumatize the canal wall membrane. If one wishes to preserve this as a plastic flap to cover the ridge and the bowl, the epitympanic recess must be thoroughly exposed. The membrana tensor must be separated from the sulcus tympanicus anteriorly, particularly at the anterior spine, so that the most anterior recess of the epitympanum just in front of the head of the malleus can be thoroughly cleaned out.

During the period of exposure of the malleus and incus the area is constantly washed out with salt solution thereby removing all epithelial débris. If there is cholesteatoma in the antrum, as is commonly found, this will have been thoroughly removed by these frequent washings. Any irregular extensions of cholesteatoma in the antrum region of the mastoid can now be smoothed out, giving a nice form to the cavity. The malleus and incus are now carefully inspected. If they are intact they are not touched. It has been my practice not to cover the exposed part of these ossicles with any part of the cutaneous canal wall flap. They stand out like a bas relief, and in a period of four years my longest case showed no evidence of necrosis of the ossicles. Four such patients, the oldest 4 years old, required nothing more than a simple removal of the accumulated epithelium every few months. The hearing in all four patients is practically normal, with not over a 10 to 15 decibel loss, the conversational range being 20 feet or better.

If after inspection there is evidence of disease of the head of the malleus, it can be removed with an instrument especially designed for this by Lempert. In these cases the incus is generally involved also, and should be removed. The gap left by its removal has closed over in all of my cases. I have been amazed at the degree of hearing that remains after the removal of the diseased head of the malleus and incus. I expected a marked drop in the hearing, and have been pleasantly surprised to find that there is only a slight drop.

There is another type of case frequently seen where there is a large posterior superior marginal perforation with cholesteatoma in the attic and antrum regions. The remaining part of the membrana tympani is quite gray and shows no evidence of inflammatory change. The epithelium from the margin of the perforation has grown down over the stapes and thence upward into the epitympanum, practically sealing off the middle ear cavity from the epitympanum. If the hearing is good or even fair, this type of case is ideally suited for the modified radical.

In these cases, at operation one generally finds that the incus has completely sloughed out, there is cholesteatoma in the antrum and attic, the head of the malleus is partly eroded but may not be. As there is no point in retaining the head of the malleus it is cut off close to the short process. The superior part of the membrano-cutaneous canal wall is cut and laid back over the facial ridge toward the bowl as in the procedure previously described.

Dr. Mosher some years ago devised the paraffin basket as a mould for the retention of the transplanted skin graft in the radical cavity. This permitted a perfect adaptation of the transplanted skin to the walls of the newly formed cavity, and shortened the after-care by many weeks. We still use it advantageously. It lends itself best for the continuous type of graft in which one large layer of skin is placed on the mould and thence into the cavity. In recent years small multiple grafts have been used. Small strips of skin are placed, one in the middle ear, one in the epitympanum, another in the bowl, with good results. If one fails to take, the others generally do.

The time for grafting has varied. Where there has been no history suggesting complications and the cavity has been cleaned out thoroughly, primary skin grafting has been used successfully. Most of us, however, prefer a graft from five to eight days after the initial operation. There are many reasons why this would be preferred by most men. In the modified radical, if one finds that the area of the tegmen and the medial wall of the epitympanum, and the antrum, tend to sprout granulation tissue, one can obviate this by planting a small graft over this area. It is done by injecting 0.5 to 1 c.c. 1 per cent novocain on each side of the vertical part of the endaural incision in front of the ear. This permits painless spreading of the wound and gives perfect exposure of the cavity. The graft is then cut, under local anesthesia, and placed in the desired area and retained with small dental pellets for two or three days.

It has been my experience that a skin graft placed in the middle ear cavity depresses the hearing below the preoperative level. Where granulations are removed from the area of the round and oval windows and the cavity grafted later, the hearing is markedly depressed. Best hearing results in the radical operation are obtained when, if conditions permit it, the mucous membrane of the tympanum proper is left untouched.

CONCLUSION

Too many otologists approach the problem of chronic suppurative otitis media with the sole idea of getting rid of the discharge, and pay little attention to the preservation of the hearing. One does not need to have a long surgical experience to accomplish both, if he will remember not to traumatize the mucous membrane in the middle ear proper, in the region of the windows.

The performance of the modified radical does require a previous experience and training. The effort expended in acquiring the necessary training, however, is well compensated by the results.

The chronic ear will be an otological problem for years to come. It is our obligation to consider the function of the ear more seriously in this condition. Let us make use of technical surgical advances afforded us, to the end that the preservation of hearing and the eradication of disease will receive equal consideration in the management of the chronic suppurative ear.

20 CHARLESGATE WEST.

REFERENCES

1. Crowe, S. J.: Diagnosis and Differential Diagnosis of Deafness, Arch. Otolaryng. 28:663 (Nov.), 1938.

2. Fowler, E. P.: Hearing Before and After Radical Mastoidectomy, Arch. Otolaryng. 26:387 (Oct.), 1937.

3. Lillie, H. I.: Discussion of Smith,[7] Laryngoscope 48:508-509 (July), 1938.

4. Kettel, K.: The Lempert Endaural, Antauricular Surgical Approach to the Temporal Bone, Arch. Otolaryng. 34:461 (Sept.), 1941.

5. Williams, H. L.: Proc. Staff Meet. Mayo Clin. 17:22-24 (Jan.), 1942.

6. Shambaugh, G.: A New Plastic Flap for Use in Endaural Radical Mastoidectomy, Tr. Chicago Laryng. & Otol. Soc., Oct. 6, 1941, reported in Arch. Otolaryng. 35:824 (May), 1942.

7. Smith, J. M.: Surgical Technique for the Conservation of Hearing in Chronic Otitis Media, Laryngoscope 48:499-509 (July), 1938.

LXIX

MASSIVE HEMORRHAGE AS AN OTOLARYNGOLOGICAL COMPLICATION*

FRED E. MOTLEY, M.D.

CHARLOTTE, N. C.

The occurrence of six sudden, fatal hemorrhages and one almost fatal hemorrhage, even though these incidents were spread over fifteen years of otolaryngologic practice, is a very sobering and thought-producing experience.

The literature has many references to deaths caused by instances of sudden, profuse, severe hemorrhages. I agree with Salinger and Pearlman,[1] who state that although several hundred cases have been collected in the literature, there are a great many more than this unrecorded. All these severe hemorrhages, in my experience, came from the large vessels of the neck, or from some tributary arising immediately from the trunk of such large vessels, so that a rapid loss of blood resulted.

The anatomy of the great vessels and the fascial planes of the neck have been discussed again and again. White and Hubert[2] state: "To Mosher, in this country, is due much of the present interest in the parapharyngeal (pharyngomaxillary) space and its inflammatory diseases." As a former Mosher house-officer, it is a source of pride and gives me a sense of reflected pleasure to note that all the articles concerning deep neck infections published in the last twenty years in American otolaryngological journals give credit or acknowledgment for their anatomical background to Mosher's famous paper.[3] Valuable contributions to the variations and unusual phases of the anatomy of the neck have been made by Barnhill,[4] Schäfer,[5] Alden[6] and Furstenburg.[7]

That the occurrence of massive hemorrhage in otolaryngological practice is an impressive happening is demonstrated by the reports of such experienced authors as Iglauer,[8] Pearlman[1] and Richards.[9]

This is further borne out by the following cases of my own:

CASE 1.—T. L. D., a listless, pale, 1-year-old boy, with bleeding from the left ear, was admitted to the hospital at 6:00 P. M. The parents stated that a

*From the Charlotte Eye, Ear and Throat Hospital.

swelling in the left side of the neck had been present for about a week. The left ear had been bleeding for the last three days, and this bleeding had been almost constant for the last two days, practically exhausting the patient.

Examination revealed a swelling of the left side of the neck, and a moderate sized, smooth, rubbery tumefaction back of the left tonsil. The pulse was of very poor quality and extremely rapid. It was my preoperative opinion that there was a lateral pharyngeal abscess, which had ruptured into the carotid sheath, and probably an erosion of the internal carotid artery.

About one hour following admission to the hospital, 125 c.c. of blood were given by indirect transfusion. Under very light ether anesthesia, a small incision was made into the retropharyngeal abscess. A tablespoonful of thin, watery pus came from the swelling and then two or three tablespoonfuls of thick blood clots were removed. An external incision was made in the left side of the neck. A large, firm, blood clot was encountered in the region of the carotid sheath. There must have been several ounces of blood distending the fascia of the carotid sheath. The usual anatomical landmarks could not be identified. There was swelling and rupture of the carotid artery wall with an aneurysm at the bifurcation of the common carotid. The common carotid was ligated, and the jugular vein was ligated above and below at the top and bottom of the incision. The facial vein was thrombosed, and necrosis had taken place at the junction of the facial vein and internal jugular. In exploring further, a gush of blood was encountered at the region of the jugular bulb. Inasmuch as no ligature could be placed in such a position, the entire wound was packed with gauze. There was no further bleeding. The leucocyte count was 50,400 at this time, and the red blood count was 3,000,000 and hemoglobin 58 per cent.

The patient died from shock about two or three hours following the operation, although he was only in the operating room about thirty minutes.

Diagnosis: Fatal massive hemorrhage from the ear and throat complicating infection of the pharyngomaxillary space.

If this patient had been seen within twenty-four to thirty-six hours after the onset of the continuous bleeding from the ear, I feel the chances for recovery would have been much greater.

Mosher[9] states that the fascial spaces around the great vessels of the neck and the pharyngomaxillary space are more potential than actual spaces until they are filled with pus. It might be added that this is also true when the pharyngomaxillary space or the carotid sheath is filled with blood; and we see even its terminal fascial extensions and ramifications when blood finds its way from the pharyngomaxillary space or carotid sheath to the external auditory canal.

Eves[10] designates three routes by which pus or blood could reach the auditory canal: (1) through the fissures of Santorini; (2) through the fibrous path at the junction of the cartilage and

the bone (present most often in children); (3) through dehiscences of the bony canal (present in a small percentage of cases).

Roberts[11] and Kaufman[12] each report a fatal hemorrhage following a retropharyngeal abscess with the complication of bleeding from the ear. Both attempted ligation. The autopsy in Kaufman's case showed death was due to erosion of the first branch of the carotid, which in turn sends a small artery to the tympanic cavity. Bleeding from around the carotid sheath extended out along this small branch toward the tympanic cavity.

Young[13] reports two cases in children and states that such infective processes in the neck attack the wall of the internal carotid artery so that an erosive aneurysm is formed with eventual perforation. Blood escaping through the perforation lodges in the pharyngomaxillary space forming a hematoma there. The blood passes upward into the parotid space, and then, following the line of least resistance, through the fissures of Santorini into the external auditory canal.

Young[13] made a search of the literature finding reports of 26 similar cases and suggested that profuse, sudden hemorrhage is frequently due to erosion of the extracranial, not the intracranial, portion of the internal carotid artery. In his summary, he stated the hemorrhages occur only in cases of erosive aneurysm and almost always in children.

White and Hubert[2] and Iglauer[8] feel that hemorrhage from the external ear or eustachian tube practically always means erosion of the internal carotid artery and calls for immediate ligation.

Salinger and Pearlman[1] add even more to the gloomy picture and report an 80 per cent mortality rate of such patients with aural bleeding. "Spontaneous hemorrhage from the ear," Salinger[14] says, "was found to be a grave symptom and was present in twenty cases and only four of these survived. Autopsy in every one of these cases showed a false aneurysm of the internal carotid artery. This symptom should be an indication for immediate ligation of the common carotid."

Frank and Abt[15] afford a little more cheerful outlook by reporting four cases of severe arterial hemorrhage from the pharynx and the middle ear, all successfully operated upon with recovery. I would assume that they ligated at the first opportunity, which must have been early in each case, and are to be congratulated upon the results.

CASE 2.—B. R., a 3-year-old boy, was referred by a pediatrician because of sore throat, fever and a swelling on the right side of the neck. He had had a sore throat at intervals for about a month.

He had large, chronically infected tonsils with a right lateral pharyngeal abscess which seemed to be located in the tissue under the tonsil and back of the posterior pillar. On the following day, a tonsillectomy and adenoidectomy were done under general anesthesia. No unusual bleeding was encountered. The acute infection subsided. With a hemostat, an enormous abscess was opened under the right tonsil and a large quantity of thick pus evacuated. The patient was kept in the hospital for two days and then permitted to go home.

He was seen four times following the operation and examined carefully because the mother stated the child was crying and fretful. He ate well but there was still an enlarged gland on the right side of the neck. The tonsil fossa healed except for a small tab of granulation tissue, about the size of a small pea, on the right side where the abscess had been opened.

Twenty days following operation and drainage of the abscess, there was a sudden pulsating gush of blood from the mouth and in a few seconds the child died. Just previous to his death, the child had eaten popcorn and candy and had been running around the room. Due to the blood splashes over the ceiling and walls of the room, I would suppose one of the carotids to have eroded.

Diagnosis: Fatal massive hemorrhage complicating a lateral pharyngeal abscess.

In this case, following the rule of some American and most of the European authors of the last ten years, the tonsils were removed at the time the abscess was drained. This same procedure has been carried out many times by our staff during the last few years with excellent results. Although I do not feel the tonsil and adenoid operation had anything to do with the final outcome here described, the fact that there was any operative procedure, certainly placed the surgeon on the defensive when explaining the death to a grief stricken family.

CASE 3.—G. D., a boy 5 years of age, was seen with a pharyngeal abscess on the right side which had opened previous to examination. The tonsils showed definite infection and because there was some glandular enlargement, tonsillectomy and adenoidectomy was done under general anesthesia.

The patient was dismissed from the hospital, doing well, two days following operation. He was readmitted to the hospital three days following operation with severe bleeding from the throat. While a large gauze sponge was held in the throat by an associate, the common carotid was ligated as rapidly as possible, the jugular vein ligated low in the neck and as high as could be reached because we were not certain as to the origin of the bleeding, except that it was from one of the large vessels of the neck which evidently had been eroded.

The white cells were 19,000; the red blood count 1,800,000; and the hemoglobin 30 per cent. A transfusion was given immediately, and repeated on the following two days. The abscess cavity had been packed with plain gauze. This

was slowly withdrawn. Two weeks later the boy was dismissed from the hospital, making an uneventful convalescence.

Diagnosis: Massive hemorrhage, with recovery, complicating a lateral pharyngeal abscess.

Because of the similarity in Cases 2 and 3, the literature is reviewed for these two cases together.

Salinger[14] gives a very comprehensive report of 227 cases taken from the literature, including 10 unreported cases of his own. Autopsy reports available in 90 of these cases showed the internal carotid was eroded 49 times, the external carotid 4 times, the common carotid 9 times, and other vessels 14 times. There was a total of 76 deaths due to erosion of arteries.

Smith,[16] Richards,[9] Brooks,[17] Speilberg[18] and Kleinfeld[19] give reports of several cases of fatal arterial hemorrhage. Jones[20] also reports several cases, in three of which the veins of the neck were the source of bleeding.

Alden,[6] Orton[21] and Beck[22, 23] give excellent anatomical surveys and discuss other complications of deep neck infections, but do not give case reports of bleeding, although Beck[23] mentions the possibility of arterial erosion.

Greenfield[24] reports 290 cases of peritonsillar infection with only three cases of severe secondary hemorrhage. Transfusions were given but the carotid was not ligated in his cases. He felt that the surgeon was often criticized because hemorrhage was attributed to injury by the knife, but actually erosive action takes place into the large vessels in the region of the abscess. This author states that autopsies show erosion takes place more often in the lingual, facial and ascending pharyngeal arteries, all branches of the external carotid; and that the internal carotid is involved less commonly. In his review of the literature for the last thirty years, he found 31 cases of severe hemorrhage, which he divided into three groups, based on the treatment employed: (1) ligation of common carotid artery with 66 per cent recovery; (2) ligation of external carotid with 44 per cent recovery; (3) conservative treatment with only 25 per cent recovery.

Hayes[25] reports two cases. The first patient recovered following compression of the carotid artery and transfusions, but with a permanent facial paralysis and a permanent limp on the opposite side, possibly due to cerebral embolus. The second case reported was a spontaneous fatal hemorrhage with almost instant death, following incision of a typical peritonsillar swelling.

Rouget,[26] Krüger,[27] Wuest,[28] D'Arcy,[29] Leegaard[30] Schäfer[5] and Meyer Zum Gottesburg[31] all report cases stressing the importance of bleeding as a complication in neck infections. All of them advise ligation.

Simonetta,[32] reporting several cases, advocates ligation of the carotid artery, although he reports severe cerebral disturbances occurring following ligation. In this author's opinion, ligation of the internal jugular vein aggravates the venous stasis and causes cerebral edema.

A transcription of a paper by Tavani[33] had an unusual group of 300 fatal cases of such hemorrhages collected from the literature, all in cases of retrolateropharyngeal abscesses, including three of his own. He felt that the internal carotid was the vessel most often attacked, except in peritonsillar abscesses, when the external carotid or its branches were most involved. The mortality rate is very high whether or not treated by ligation. This article goes on to state that in the pathogenesis of such hemorrhages, the chief factors are the invading organism and its virulence, the incompleteness or lateness of intervention, and the resistance, both local and general. This Italian author feels that small hemorrhages should always be taken as a sign of threatened large, profuse bleeding; that the surgeon should be ready for ligation and, even though prepared, there may be no opportunity, owing to the acute character of the bleeding.

Barnhill[4] states that thrombosis of internal jugular vein or its branches may take place in any deep infection of the neck located along the carotid sheath. These veins are involved through an extending septic process. He feels that in most cases where there is necrosis of the carotid artery or the walls of the jugular vein, there has been neglect in allowing the case to go so long without drainage and that this is the primary background of fatal hemorrhage which often follows. He feels that the mortality should be reduced in these cases of hemorrhage from 50 per cent to at least 25 per cent. Barnhill[4] feels that every deep abscess of the neck is a case for surgery; that medication, poultices, and other general local treatment only cause delay; that these are not curative, and their continued use only invites complication; and once pus forms in the deep tissues of the neck, it must be evacuated.

Pond[34] has a cleverly written paper with the usual background of anatomic studies referring to Mosher,[3] Beck[22, 23] and others. He reports several hemorrhages following incision of lateral pharyngeal abscesses. He criticizes himself for two mistakes: first, for wait-

ing too long; and, second, for not opening this type of abscess externally, instead of through the mouth.

Lifschutz[35] reviewed the literature and reported one case of his own. It was his opinion that such hemorrhage was more common in children and that ligation of the common carotid was the only treatment. Of the 23 cases considered in his paper, there were 19 deaths and four recoveries, three of which recovered after ligation, one without ligation. He makes an interesting historical comment, that Liston in 1843 was probably the first to show that abscess of the cervical region could communicate with the carotid artery.

Lifschutz[35] also states that the presence of an anomaly or vascular disease may facilitate the erosion of vessels, but there is no reported case where infection has complicated a known anomaly or vice versa. He believes the slightest sign of hemorrhage associated with retrolateropharyngeal abscess calls for the ligation of the internal carotid artery. He thinks there may have been a few cases in the literature where injury from a stiff drainage tube was a factor causing erosion of the artery wall.

Salinger and Pearlman[1] report 10 cases from the records of Michael Reese Hospital and the largest analysis of cases in the American literature. The results show those surgeons who ligated the carotid, especially the common carotid artery, seem to have had the best results. The autopsy reports compiled in this paper show the internal carotid is attacked in over one-half the cases, and that the internal jugular is seldom involved except in large sloughs in the neck produced by scarlatina. This paper stands out in its discussion of percentage of risks and complications in ligation of the carotids, and also in its detailed and careful recording of the literature.

Iglauer[8] wrote seven years ago on surgical approaches to the neck and mediastinum and again gives his idea of the relative importance of these neck infections by a reported case of serious bleeding in a retropharyngeal abscess case that recovered after many complications. This case he lists as "my most unusual case". He tersely states, "Never ignore bleeding, ligate!"

White and Hubert[2] feel that the common carotid should be ligated slowly, in minutes, not seconds, thereby avoiding complications. There is repeated emphasis in their article on the parapharyngeal relations of the ninth, tenth and twelfth cranial nerves, and the sympathetic trunk and its superior cervical ganglion.

Brunner[36] recently reported 20 cases of pharyngeal sepsis from the Vienna Polyclinic, but no cases dealing with hemorrhage were considered.

In a recent pediatric consideration of abscess of the throat by Deering and Brennerman,[37] the authors advocate delay until there is "not only fluctuation, but advanced fluctuation." They recognize that hemorrhage is reported by other authors but they did not encounter it in their cases and permitted over 50 per cent of their retropharyngeal abscesses and other patients with throat abscesses to drain spontaneously. With regard to complications, they felt that: "Luck seems to have been with us in our series." I thoroughly agree with them, judging not only from my own experience but that of most other otolaryngologists writing on the subject.

Richards[9] has written a careful consideration of retropharyngeal abscess and reports 162 cases. He considers three hemorrhage cases and gives a percentage chance of hemorrhage encountered as 7.5 per cent.

Carmack[38] devoted a long article to anatomical discussions and references as to the possible abnormalities of the blood supply of the neck. All unusual anomalies of vessels in the neck are considered.

Rosenberg and Berke[39] and Havens[40] recognize the risk of severe hemorrhage as a complication of retropharyngeal abscess. Havens[40] also reports five cases of death due to hemorrhage following surgery on the throat, but only one case followed infection in the neck. Like myself, he was unable to obtain post-mortem examinations. Schaeffer is quoted in Salinger and Pearlman's[1] article as reporting three fatal cases of hemorrhage due to the presence of an aberrant internal carotid artery during tonsillectomy. While the possibility of aberrant vessels and aneurysms must be kept in mind, I feel it is a much more important problem in elective surgery of the throat, such as tonsillectomy, than it is as a factor in hemorrhage occurring in infections in the pharyngeal region.

Most of the German, Italian, English (see abstract references) and many American authors, advocate tonsillectomy as a part of the treatment for the better drainage of peritonsillar abscesses. Some of these men consider tonsillectomy as not only a curative but as a prophylactic measure so far as complications are concerned. I do not feel the tonsil surgery in Case 2 and 3 had anything to do with the hemorrhage. We have operated upon many such cases with no untoward results.

CASE 4.—E. L., a 6-year-old boy, was admitted to the hospital with a history of having had marked difficulty in breathing for the last few hours. He had had a severe sore throat, diarrhea and bloody stools for a week previous to admission.

There was excoriation of the external nares. The entire mucosa of the nose, nasopharynx, pharynx and larynx was very edematous, with a white exudate covering the pharynx and fauces. Immediate tracheotomy was done because of the dyspnea. Clinically it seemed that we were dealing with an acute streptococcic infection and this was confirmed by the laboratory examination. Sulfanilamide was given. Light X-ray therapy was started for the adenitis and cellulitis of the neck. The white blood count on admission was 46,200; the hemoglobin was 61 per cent; and the red blood cells were 4,080,000.

The child seemed very toxic and during the next ten days, in spite of four blood transfusions of 200 c.c. each, the red blood count dropped to 3,840,000 and the hemoglobin to 58 per cent. The tracheotomy wound sloughed over a large area and there was a deep ulcer on the anterior surface of the neck.

Fifteen days after entrance to the hospital, there was bleeding from the tracheotomy wound (probably from one of the thyroid vessels) and into the tracheotomy wound so that the patient coughed up large amounts of bright blood. There was a sudden, marked swelling which appeared in the right upper neck. After exposure and drainage of the right pharyngomaxillary area, the right common carotid artery was ligated. A tracheotomy tube was reinserted, the tube being packed off from the rest of the wound with gauze. All bleeding ceased following operation. Seven hundred cubic centimeters of blood was given during and immediately following the operation.

On examination a small break was seen in the mucous membrane at the base of the right tonsil and possible bleeding from a large vessel through this area was considered.

Five days later, there was an alarming hemorrhage coming from both the mouth and tracheotomy wound. At this time there was severe bleeding coming from the opening at the bottom of the right tonsil fossa. This was packed with a large amount of gauze. It was felt that this bleeding might have come from around the jugular bulb because the common carotid had been ligated on this side. The patient was again given 500 c.c. of blood. A feeding tube was placed through the nose into the stomach so that the throat would be placed at rest. The patient rallied reasonably well.

Blood transfusions were given on three successive days and the red blood count came up to over 4,000,000 and hemoglobin to over 70 per cent. However, because of severe pyelitis and nephritis, it became necessary to discontinue the sulfanilamide, and the temperature, which had never reached normal, began to go up again.

The laboratory reported cultures showing hemolytic streptococci predominating with occasional colonies of staphylococci. There were repeated severe external hemorrhages coming from both the right internal maxillary vein and from the lingual vein. The last few days before the patient's death, nearly five weeks following admission, there was some bleeding from low in the neck probably from the stump of the jugular vein. There was a great deal of blood in the urine for the

last few days before death. Zinc peroxide was used for dressings and this seemed to clean up the wound a great deal better than other drugs.

This infection followed the great vessels of the neck on both sides and practically destroyed the muscular tissue surrounding the trachea and larynx. The slough of the area around the right carotid sheath exposed the base of the skull in the pharyngomaxillary region and the musculature of the neck, the digastric muscle standing out well, both anterior and posterior bellies.

Diagnosis: Repeated massive hemorrhage with fatal termination following bilateral parapharyngeal infection. Originally this began as a widespread streptococcic infection involving all the mucous membrane of the upper respiratory tract, and was complicated by a cellulitis of the neck that resulted in extensive slough and destruction of tissue.

The care of this boy, who was one of a share-cropper's 11 children, exhausted both the surgical and nursing staff of the hospital, and almost exsanguinated our local supply of Group 4 Red Cross donors. Although there was overwhelming toxemia from the time this patient was first seen, I feel the necessary use of sulfanilamide over several weeks added to his kidney damage. If only one side of the neck had been involved, I think this boy's condition might not have been so hopeless surgically. However, with the extensive sloughing after ligation of the right carotid, a tracheotomy wound with a superficial slough extending from the hyoid bone to the suprasternal notch, and a necrosis and skeletonization of the deep structures and vessels on the left, the problem of where to pack and ligate became indeed serious.

Possibly a well-nourished patient in better general condition might have been able to handle such an infection from the onset. Perhaps the earlier use of one of the sulfa drugs, plus repeated small transfusions at the onset before hospitalization, might have prevented a fatal outcome. It was the unfortunate story of our defense against an overwhelming streptococcic neck infection being "too little and too late". It seemed that the use of zinc peroxide helped clean the wound and handled the foul sloughing areas better than any other means of dressing at our disposal. I would try its use earlier if given another opportunity.

Willemin-Clog[41] has written concerning a necrotic variety of angina which occurs either as a complication of scarlatina or as a complication of severe streptococcic infection. He described a sloughing area of the pharynx which extended into the parapharyngeal space. If there was glandular involvement and phlegmon, large sloughs occurred. He stated the end results of such cases to be cicatrization and scarring or perforation of a large vessel of the neck. The prognosis was given as grave and the mortality up to 75 per

cent, with death chiefly coming from hemorrhage or sepsis. The possibility of a prophylactic ligation of the large vessels was not discussed in the paper.

CASE 5.—Mrs. W. S. H., 54 years of age, came in for an examination because she had had several severe sore throats during the last few years. Her ear, nose and throat examination was essentially negative, except for submerged, infected tonsils. There were matted, slightly enlarged cervical glands on both sides of the neck. A medical consultant reported that there was some thyroid disturbance and a moderate hypertension.

A tonsillectomy was done under local anesthesia. There was not the usual local hemostatic effect from the injection of novocain and adrenalin and there was brisk bleeding from both tonsil fossae after removal of the tonsils. A solution of 3/4 per cent novocain (to which was added two drops of adrenalin to the dram) was used.

The bleeding was controlled with one suture in the center of each fossa and several ties on the small vessels. There was immediate swelling and extravasation of blood into both the posterior and anterior pillars and the soft palate. Except for this apparent bleeding into the tissues, there was no further loss of blood. I thought at the time that this bleeding might be due to an unusual elevation of her blood pressure; but, on investigation, it was found to be normal.

It was my opinion that this bleeding was due to very fragile capillaries with extravasation of blood into the tissues; because all vessels of any size were promptly ligated at the time of operation and there was no bleeding of any consequence following the operation.

The patient had a very stormy time with increasing swelling and complete lack of use of the palate due to edema. There was a secondary infection of the tissues of the throat, which were devitalized and offered very little resistance, on account of the swelling and interference with the circulation. There was a thick, foul, tenacious exudate covering the uvula, nasopharynx, soft palate, and even extending across the base of the tongue.

Repeated cultures showed streptococcus and staphylococcus abundant. No diphtheria and no Vincent's organisms were found. (Although Vincent's organisms were not found, it is my opinion that in these cases there is often an underlying Vincent's infection and, clinically, they should be treated as a potential Vincent's infection.)

Neoarsphenamine and thiobismol were given. Sulfanilamide was used for a few days and this was changed to sulfapyridine. There was no further loss of blood following the operation. There was a very foul odor and sloughing was present in each tonsil fossa. Hot throat irrigations were given several times a day and both fossae were cleaned daily with peroxide.

Blood transfusions of 300 c.c. each were given on the seventh, ninth, eleventh and fourteenth days after operation. Laboratory report, fifteen days after the operation showed: white blood cells, 9,500; red blood cells, 3,760,000; hemoglobin, 12 gm. or 78 per cent (Haden-Hausser). Differential: p-68%; L-26%; L. M.-1%; T-3%; E-2%; B-none.

Additional blood transfusions were given on the sixteenth and eighteenth days after operation. A subsequent laboratory report showed: white blood cells, 5,200; red blood cells, 4,008,000; hemoglobin, 13 gm. or 84 per cent.

The patient was examined and each tonsil fossa found to be clean. There was little or no evidence of infection or slough and there was early formation of healthy granulation tissue.

Twenty-five days following operation, the patient had a sudden, explosive type of hemorrhage with almost instant death. While I was not at the hospital at the time of her death, I arrived and examined the patient within four or five minutes after the onset of her bleeding, and it was impossible to determine from which side the bleeding occurred.

Diagnosis: Bilateral pharyngomaxillary infection following local tonsillectomy. Extensive sloughing and necrosis of the pharynx and palate with fatal massive hemorrhage.

Soubiron and Emiliani[12] report a fatal case of parapharyngeal infection and swelling following tonsillectomy. The authors feel that probably the cause was penetration of the local anesthetic into the parapharyngeal space. The abscess was followed by ulceration of the arteries of the neck and severe hemorrhage. There was a jugular thrombophlebitis and a cerebral abscess.

Seltz[13] writes of a patient who had a large pharyngeal abscess develop one week after tonsillectomy. The abscess was opened through an external incision in the neck with recovery.

Shapiro[14] has a well-written article, with a summary of the literature to date, reporting 30 cases of his own. He states that deep cervical infection following operation is almost exclusively a complication of local tonsillectomy. The pathogenic bacteria were found to be the usual flora of the mouth and throat. He considers the possible sources of infection to be: (1) tonsillar tissue; (2) contamination of the needle or solution; (3) contaminated instruments; (4) thrombosis of the tonsillar veins by way of the ascending pharyngeal, the anterior facial, the posterior palatine to the internal jugular or to the pharyngeal plexus; (5) by way of lymphatics, such as, a lymphanginitis producing a very severe phlegmonous infection without any any tendency to suppuration.

White[15] reported a unilateral pharyngomaxillary infection which began three days following a local tonsillectomy. A later external incision was necessary with through-and-through drainage. His patient recovered.

Case 6.—L. J., a cotton mill worker, aged 37 years, was admitted to the hospital with a history that ten days previously he had had a tearing sensation in the right side of the neck while at work. There was severe pain and prostration

for a few minutes which necessitated his lying down on the floor. However, he kept on working for a few days and then stayed at home for several days under the care of his family physician. He had been unable to swallow even fluids for the preceding four days and there was marked hoarseness. The pain in the neck was still severe and he had had quite a high fever.

On examination this man appeared to be seriously ill. He was very pale with an anxious facial expression and evidently in pain. His ear, nose and throat examination was essentially negative except for large, infected tonsils and a paralysis of the right side of the larynx. There was a large, diffuse, smooth swelling of the right side of the neck across the thyroid region extending down to the clavicle. No thrill or bruit was present. Infection of the mediastinum or an aneurysm was considered in the differential diagnosis. The white blood count was 23,000; the red blood count was 3,600,000; and hemoglobin, 71 per cent. The Kline test was two plus. X-ray studies of the neck disclosed a large, smooth, shadow in the right upper mediastinum. Because of serious dehydration, he was given large amounts of glucose by rectum and intravenously for twenty-four hours before operation.

Two days later exploration of the neck was performed. A preliminary laryngoscopy and esophagoscopy were done by my associate, Dr. V. K. Hart, with negative findings. A bronchoscope was left in the trachea in order to avoid tracheal obstruction or respiratory difficulty. Under pentothal intravenous anesthesia plus novocain infiltration, an incision was made along the posterior border of the sternocleidomastoid. The distorted tissues and markedly enlarged jugular made this approach virtually impossible. The sternocleidomastoid muscle was retracted laterally and an approach made anterior to the muscle. In dissecting with the finger in order to reach the mediastinum, foul pus was encountered, and about 6 or 8 ounces were removed. An incision was made on the left side of the neck and a drainage tube introduced under the trachea, allowing through-and-through drainage. A large, fenestrated, soft, rubber tube was placed on the right side down into the anterior mediastinum.

Sulfapyridine was given throughout his illness. Bacteriological studies made of the pus obtained at operation, including anaerobic cultures, showed only Staphylococcus albus.

The patient's temperature was 106°, and his condition was critical immediately after operation. However, after further intravenous glucose, the temperature dropped. The drainage tubes were changed and there was copious drainage of very foul-smelling pus. The temperature remained almost flat. Two hundred ounces of fluids and liquid foods of high caloric value were taken during the day following operation. The patient's condition improved rapidly, and a blood transfusion of 500 c.c. was given.

The X-rays which were taken following operation showed the drainage tube extending into the upper mediastinum.

On the seventh and ninth days after operation, the patient had two moderate and one severe hemorrhage. Although the exact amount of bleeding could not be determined, it was sufficient to soak the bed and mattress and to cover part of the floor of the hospital room. The pulse was very rapid. The hemorrhage was controlled by packing and a transfusion was given the following morning. The neck was reopened by my partner, Dr. V. K. Hart, because of my absence from the hospital. A large hole was found in the internal jugular vein and the common

carotid artery was covered by a sloughing, necrotic mass. It was not deemed safe to put a ligature around it or do any unnecessary dissection. The jugular bulb was packed off with gauze, another pack was placed around the lower portion of the vein, and a third pack was placed around a very soft rubber drain into the upper mediastinum. The incision was not closed. The patient was immediately transfused with 500 c.c. of blood.

An additional 1500 c.c. of blood was given during the next seventy-two hours. He had only one small hemorrhage during that time. Two intravenous administrations of neoarsphenamine were given because of some gingivitis and because of the two plus Kline. The sulfapyridine was reduced to 20 gr. daily.

Death followed an exsanguinating hemorrhage, sixteen days after admission to the hospital. Repeated blood cultures showed no growth.

Diagnosis: Massive fatal hemorrhage following suppurative mediastinitis of unknown origin.

No post-mortem examination was obtained although it would have been particularly instructive in determining the etiology. The following possibilities must be kept in mind: (1) The sudden onset suggests a small bronchial tear, following a cough, with a subsequent emphysema. The resulting infection came slowly, walling off, and forming the mediastinal abscess. (2) Esophageal injury by a foreign body, of temporary sojourn in the esophagus, which was dislodged and forgotten. (3) Rupture of an old caseous gland with resulting secondary mediastinal infection and abscess formation. Furstenburg and Yglesias[7] consider mediastinitis and give special consideration to the anatomy. There is little direct reference in the literature at my disposal regarding hemorrhage following mediastinitis. However, the same rules of pathology would apply as with infections higher in the neck and the surgical attack would be even more hazardous and difficult.

Case 7.—This 15-year-old girl was brought into the hospital at six o'clock in the afternoon. There was a history of intermittent chills and fever for nearly a month with more severe chills for the last week. Although she had been troubled with a foul, discharging ear since infancy, the family physician had been giving her quinine in large doses for a period of several weeks for supposed malaria. She had been taken in consultation to a more thoroughly trained general practitioner who felt that the ear might have some bearing on her case and sent her into the hospital for study.

The examination was essentially negative except for destruction in the upper half of the drum and the entire attic region of the right ear. There were small pads of granulation tissue in the middle ear, some cholesteatomatous material, and a very foul discharge. The temperature was about 101.5°. It was planned (because there seemed to be no emergency about this girl's condition) to have X-rays made of the mastoid and a complete laboratory study done the following morning. At that time, if her chills and fever were felt to be coming from the otorrhea and evident chronic mastoiditis, a radical mastoid was to be done. However, at about

eleven o'clock during the night the girl's mother, who was staying in the room, rang the bell. The bed, mattress, and part of the floor were found soaked with blood from a profuse, rapid hemorrhage coming from the involved ear. The nurse attempted to arrest the hemorrhage with cotton pledgets while I was called on the telephone. On arrival, I found conditions as described and the patient dead, undoubtedly from exsanguination. There was no history of bleeding previous to the sudden fatal hemorrhage.

Diagnosis: Fatal hemorrhage complicating right chronic otitis media and chronic mastoiditis.

If this girl had been operated on immediately upon admission to the hospital, she would have had a better chance for her life. However, it has never been my custom to classify as an emergency a patient with a history of a discharging ear of long standing with symptoms of a month or more possibly referable to the ear.

There are few reported cases of fatal hemorrhage from the ear. Benech[16] reports an uncontrollable hemorrhage from the lateral sinus during acute otitis media and mastoiditis in the desquamation stage of scarlatina in a 12-year-old girl.

Roux[17] also reports death from a sudden spontaneous hemorrhage from the internal carotid artery in a case of chronic mastoiditis in a Senegalese soldier.

Eadie[18] also reports a severe but not fatal case of hemorrhage from the ear in a case of acute suppurative otitis media and mastoiditis in a 5½-year-old girl. The internal, external, and even the common carotid arteries were ligated. He presumes it to have been due to an erosion of the internal carotid artery near the mastoid.

SUMMARY

1. Seven cases of massive hemorrhage are reported with one recovery and six fatalities.

2. A review of the available literature shows fatal hemorrhage to be a fairly commonly reported complication of deep neck infections.

3. Bleeding from the throat associated with peritonsillar or pharyngomaxillary infection is a danger sign that calls for immediate ligation.

4. Bleeding from the ear, if associated with throat or neck infection and swelling, calls for immediate ligation of the carotid artery.

5. The experience gained in this small series would be that tonsillectomy during peritonsillar infection does not prevent hemorrhage.

106 W. Seventh St.

BIBLIOGRAPHY

1. Salinger, Samuel, and Pearlman, S. J.: Hemorrhage From Pharyngeal and Peritonsillar Abscesses, Arch. Otolaryng. 18:464, 1933.

2. White, F. W., and Hubert, Louis: Parapharyngeal Hemorrhage; Diagnosis and Treatment, Arch. Otolaryng. 29:1, 1939.

3. Mosher, A. P.: The Submaxillary Fossa Approach to Deep Pus in the Neck, Tr. Am. Acad. Ophth. & Otolaryng. p. 29, 1929.

4. Barnhill, J. F.: Deep Abscesses of the Neck, Am. J. Surg. 42:207, 1938.

5. Schäfer, R.: Aneurysm of Carotid With Fatal Bleeding in Retropharyngeal Abscess, Klin. Wchnschr. 17:135, 1938; abst. in Otol., Laryng., Rhin. & Ophth. Internat. Abst. J. 3:142, 1937-38.

6. Alden, A. M.: Review of Some of the More Important Contributions to the Literature on Neck Infections During the Past Ten Years, Laryngoscope 49:423, 1939.

7. Furstenburg, A. C., and Yglesias, Luis: Mediastinitis, Arch. Otolaryng. 25:539, 1937.

8. Iglauer, Samuel: Surgical Approaches to Deep Suppuration in the Neck and Posterior Mediastinum, Arch. Otolaryng. 21:707, 1935; An Unusual Case of Retropharyngeal Abscess, Annals of Otology, Rhinology and Laryngology 50:215, 1941; Anatomico-pathologic Studies of Retropharyngeal Abscess, Arch. Otolaryng. 33:31, 1941.

9. Richards, Lyman: Retropharyngeal Abscesses, New England J. Med. 215:1120, 1936.

10. Eves, C. C.: Retropharyngeal Abscess Discharging Through the Right Ear, Tr. Coll. Phys. Philadelphia 48:339, 1926.

11. Roberts, E. R.: Fatal Idiopathic Pharyngeal Hemorrhage, Laryngoscope 40:807, 1930.

12. Kaufman, Aaron: Fatal Case of Pharyngeal Hemorrhage, Laryngoscope 41:826, 1931.

13. Young, N.: Bleeding From the Ear as a Sign of Leaking Aneurysm of the Extracranial Portion of the Internal Carotid Artery, J. Laryng. & Otol. 56:35, 1941; abst. in Otol., Laryng., Rhin. & Ophth. Internat. Abst. J. 6:311, 1941.

14. Salinger, Samuel: Hemorrhage From Pharyngeal and Peritonsillar Abscess, Laryngoscope 44:765, 1934.

15. Frank, Ira, and Abt, A. F.: Hemorrhages Complicating Deep Pharyngeal Infection, J. Pediat. 9:68, 1938.

16. Smith, J. E.: Retropharyngeal Abscess With Reference to Abnormally Large Percentage of Adult Cases, ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY 49:490, 1940.

17. Brooks, E B..: Acute Retropharyngeal Abscess, Laryngoscope 41:671, 1931.

18. Speilberg, William: Report of Two Cases of Pharyngeal Hematoma With Fatal Hemorrhage From Retropharyngeal Abscess, Laryngoscope 44:511, 1934.

19. Kleinfield, Lewis: Hemorrhage From the Throat, Laryngoscope 48:253, 1938.

20. Jones, A. C.: Report of Three Cases of the Rupture of Abscesses Into the External Auditory Canal Other Than That of the Parotid, ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY 43:624, 1934.

21. Orton, H. B.: Deep Infections of the Neck, ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY 47:48, 1938.

22. Beck, A. L.: Study of Twenty-Four Cases of Neck Infection, ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY 42:741, 1933.

23. Beck, A. L.: Deep Neck Infection, ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY 48:940, 1939.

24. Greenfield, S. D.: Ligation of External Carotid Artery for Uncontrollable Hemorrhage in Case of Peritonsillar Infection, Laryngoscope 43:929, 1933.

25. Hays, Howard: Serious Throat Infections, Laryngoscope 42:212, 1932.

26. Rouget, J.: Ligation of Blood Vessels in Pharyngeal Hemorrhages, Arch. Otolaryng. (Paris) p. 767, 1939; abst. in Otol., Laryng., Rhin. & Ophth. Internat. Abst. J. 5:114, 1939-40.

27. Krüger: Fatal End of a Peritonsillar Abscess, Minutes of Thirtieth Session of the Society of Lower Saxonian Otorhinolaryngologists in Rostock, Hals. & Arzt. 49:146, 1939; abst. in Otol., Laryng., Rhin. & Ophth. Internat. Abst. J. 5:299, 1939-40.

28. Wuest: Fatal Postoperative Bleeding After the Opening of Peritonsillar Abscess, Hals. & Arzt. 44:162, 1937; abst. in Otol., Laryng., Rhin. & Ophth. Internat. Abst. J. 2:253, 1937.

29. D'Arcy, T. N.: Case Demonstrating an Unusual and Fatal Ending of Tonsillar Sepsis, J. Roy. Nav. M. Serv. 23:346, 1937; abst. in Otol., Laryng., Rhin. & Ophth. Internat. Abst. J. 3:81, 1937-38.

30. Leegaard, T.: Retropharyngeal Abscess With Erosion of the Internal Carotid, Death, Norsk. Mag. Laegevid. 98:1421, 1937; abst. in Otol., Laryng., Rhin. & Ophth. Internat. Abst. J. 3:81, 1937-38.

31. Meyer Zum Gottesburg, A.: Spread of Peritonsillar Suppuration in the Peripharyngeal Space; Peritonsillar Abscess and Fistula of the Auditory External Meatus, Arch. f. Ohren. Heilk. 144:222, 1938; abst. in Otol., Laryng., Rhin. & Ophth. Internat. Abst. J. 3:284, 1937-38.

32. Simonetta, B.: Sequelae of Ligation of Common Carotid Alone or Together With Simultaneous Ligation of Internal Jugular Vein, Valsalva 12:117, 1936; abst. in Otol., Laryng., Rhin. & Ophth. Internat. Abst. J. 2:10, 1936-37.

33. Tavani, E.: Erosion of the Large Vessels of the Neck in Peripharyngeal Surgery, Valsalva 13:105, 1937; abst. in Otol., Laryng., Rhin. & Ophth. Internat. Abst. J. 2:220, 1936-37.

34. Pond, C. W.: A Study of Ten Cases of Deep Infection of the Neck, Annals of Otology, Rhinology and Laryngology 48:1055, 1939.

35. Lifschutz, Jacob: Fatal Hemorrhages Resulting From the Extension of a Retropharyngeal Abscess to the Deep Vessels of the Neck, Arch. Otolaryng. 14:149, 1931.

36. Brunner, Hans: Infections of the Parapharyngeal Space, Arch. Otolaryng. 31:597, 1940.

37. Deering, William, and Brennerman, Joseph: Acute Abscess of the Throat, J. A. M. A. 118:1171, 1942.

38. Carmack, J. W.: Aberrant Internal Carotids and Their Relation to Surgery of the Pharynx, Laryngoscope 39:707, 1939.

39. Rosenberg, Lester, and Berke, Morris: The Complications of Retropharyngeal Abscess, J. Pediat. 4:764, 1934.

40. Havens, F. Z.: Severe Hemorrhage Secondary to Retropharyngeal Abscess, Am. J. Dis. Child. 58:1256, 1939.

41. Willemin-Clog, L.: Cases of Necrotic Angina: Diagnosis and Treatment, Bull. Soc. pediat. (Paris) p. 429, 1936; abst. in Otol., Laryng., Rhin. & Ophth. Internat. Abst. J. 21:133, 1936-37.

42. Soubiron, N. V., and Emiliani, C. M.: Rare and Severe Postoperative Complication of Tonsillectomy, Rev. Asoc. Med. Argent. 52:721, 1938; abst. in Otol., Laryng., Rhin. & Ophth. Internat. Abst. J. 3:344, 1937-38.

43. Seltz, Samuel: Retropharyngeal Abscess Complicating Tonsillectomy, With External Approach, Laryngoscope 41:341, 1931.

44. Shapiro, S. L.: Deep Cervical Infection Following Tonsillectomy, Arch. Otolaryng. 11:701, 1930.

45. White, J. W.: Infection Involving the Pharyngomaxillary Space, Arch. Otolaryng. 12:248, 1930.

46. Benech: Hémorrages incoercibles du sinus latéral au cours de la scarlantine, Ann. otolaryng. p. 19, 1936; abst. in Otol., Laryng., Rhin. & Ophth. Internat. Abst. J. 1:296, 1936.

47. Roux: Tuberculosis of the Mastoid; Death From Sudden Hemorrhage From the Internal Carotid, Rev. Laryng., Otol. 59:103, 1938; abst. in Otol., Laryng., Rhin. & Ophth. 3:191, 1938-39.

48. Eadie, C. M.: Acute Suppurative Otitis Media and Mastoiditis With Severe Hemorrhage From the Ear, Presumed Erosion of the Internal Carotid Artery, Ligation of Internal, External and Common Carotid Arteries, M. J. Australia 2:9, 1941; abst. in Otol., Laryng., Rhin. & Ophth. 6:332, 1941.

ABSTRACTS OF FOREIGN LITERATURE

1. Zollner, F.: Tonsillectomy or Incision in Peritonsillar Abscess? Ztschr. f. Hals.-Nasen. u. Ohrenh. 35:509, 1934; abst. in Arch. Otolaryng. 21:226, 1935.

2. Linck, A.: Simple Incision and Secondary Tonsillectomy; and Tonsillectomy in the Course of Peritonsillar Abscess, Ztschr. f. Laryng., Rhin., Otol. (Teill: folia Otolaryng.) 25:79, 1934; abst. in Arch. Otolaryng. 21:227, 1935.

3. Tonndorf, T.: Tonsillectomy During the Acute Stage of Peritonsillar Abscess, Ztschr. f. Hals- Nasen- u. Ohren. 36:400, 1934; abst. in Arch. Otolaryng. 21:501, 1935.

4. Shroeder, R.: Some Remarks on Suppuration in the Parapharyngeal Space, J. Laryng. & Otol. 51:631, 1936; abst. in Arch. Otolaryng. 27:490, 1938.

5. Shroeder, R.: Surgical Treatment of Peritonsillar Inflammation, J. Laryng. & Otol. 51:574, 1936; abst. in Arch. Otolaryng. 27:489, 1938.

6. Thacker, Neville, W. S.: Radical Treatment of Peritonsillar Abscess, J. Laryng. & Otol. 51:650, 1936; abst. in Arch. Otolaryng. 27:489, 1938.

LXX

THE CLINICAL EXAMINATION OF THE HARD OF HEARING*

WERNER MUELLER, M.D.

BOSTON

In 1940 the Winthrop Foundation for the Study of Deafness was established at the Massachusetts Eye and Ear Infirmary. As one of the workers of the team, the writer was assigned to carry out the clinical ear, nose and throat examinations on all patients that were to be studied. Only such patients were considered for study whose deafness was not due to any obvious ear pathology. The purpose of this paper is to describe in detail the procedures that were carried out in the clinical examination of more than 500 patients ranging in age from 2½ years to 50 years. Originally the age limits had been set at 3 and 30 years respectively, but as the work progressed numerous exceptions had to be made. The great majority of patients have, so far, been seen only once. The findings resulting from each examination were taken down in detail regardless of their known or unknown significance and incorporated in the patient's record.

In the following the various headings are given in more or less tabular form along with the principal features that seemed pertinent. A critical discussion of the actual findings will not be made a part of this report. The study of deafness as carried out under the auspices of the Winthrop Foundation comprises many phases of which the clinical examination is only one. Not until all the data can be brought together will a clinical evaluation of the physical ear, nose, and throat findings be attempted.

The condition of the auricle and the external auditory meatus was remarked upon only when they showed congenital or pathological abnormalities. The presence or absence of the cough reflex and of cerumen were at first disregarded, but since their absence is by some authorities considered to be significant in the diagnosis of otosclerosis they were given, somewhat reluctantly, a place in the scheme of the examination.

*From the Department of Otolaryngology of the Massachusetts Eye and Ear Infirmary.

The tympanic membrane was carefully examined for thickness, color, luster, light reflex, general appearance and position of the malleus, and the discernibility of the long crus of the incus, the tendon of the stapedius muscle, and the niche of the round window. The three latter were grouped under the term "middle ear structures". Notice was further taken of regional retractions, scars, open or healed perforations, and calcareous deposits. The mobility of the drum membrane and the malleus was tested by means of the Siegle-Bruenings pneumatic otoscope. Finally the Gelle test was performed in the following manner: the tip of a Politzer bag was covered by a short piece of pure gum tubing and snugly inserted in the external auditory meatus so that any escape of air was practically impossible. A C-256 fork of large size was struck, its stem placed against the bottom of the Politzer bag and the latter alternately compressed and released with the other hand. With this technique the fading away and returning of the sound intensity of the tuning fork are so remarkable that the patient needs but little coaching, very often none whatever, to elicit his reaction. The writer was able to prove to his satisfaction that the change of air pressure within the bag itself does not noticeably change the degree of sound conduction.

The eustachian tubes were examined for patency by Politzer's method before the nasal mucosa had been shrunk. Closure of the nasopharynx against the oropharynx was effected by the following means and in the order given:

1) The patient was made to say "kay-kay-kay" and on the second or third "kay" sound the Politzer bag was compressed. Leakage of air in either nostril was carefully avoided.

2) If the tubes did not open on the "kay" sound, the patient was asked to swallow air. In a man the exact moment for compression of the air bag could readily be determined by merely watching the prominence of the thyroid cartilage: when the latter moved upward, the bag was compressed. In women and children the examiner's eye was kept well on the region of the thyroid cartilage.

3) If the swallowing of air was not effective, the patient was given water to swallow.

The patient was, of course, always questioned as to the result of the inflation, but in order to avoid misunderstandings an auscultation tube was invariably used to help determine the condition of the eustachian tube. The results were then noted as, e.g., "E.T.S. opens readily subjectively", "E.T.D. opens with difficulty subjectively", "E.T.U. opens readily subjectively and objectively", etc.,

according to the patient's statement and the examiner's observation. Failure of the tubes to open was often apparently due to postoperative mutilation of the soft palate. If the tubes refused to open by Politzer's method, catheterization was resorted to, but only after the examination of the nasopharynx, nose and throat had been completed since it involved shrinking and local anesthesia of the mucosa of those regions.

In examination of the nose the color, thickness and moisture of the mucosa were noted, also the size and shape of the turbinates and the septum. The mucosa was then shrunk in order to render the deeper portions of the nasal cavities more easily visible. If the history and the conditions noted warranted it, an X-ray examination of the sinuses was asked for. Patency of the choanae and the appearance of the posterior nasopharyngeal wall were checked, after shrinking, by looking through the inferior meatuses and having the patient say "kay", since at that moment the soft palate rises and can often readily be seen through the patent inferior meatuses. Under normal conditions the light reflex on the posterior nasopharyngeal wall does not change much if at all.

If the inferior meatuses of the nose were wide enough to admit the passage of the Holmes nasopharyngoscope this was used to examine the eustachian tube orifices. Local anesthesia was always applied as follows: A wooden applicator of the common variety was laid upon a strip of cotton 3 inches long, ½ inch wide and about ⅛ inch thick. By properly twisting the applicator, about one-half of its length was then completely covered by cotton. This end was squeezed out of 2 per cent pontocaine and then inserted in the inferior meatus well on the floor of the nose. A thoroughly anesthetized "tunnel" was thus created which was large enough to admit the nasopharyngoscope without the slightest discomfort to the patient. The patient was told that he would "feel pressure but no pain". Incidentally, "vocal anesthesia" was found to be very useful during all procedures that were the least bit strenuous or unpleasant to the patient. The right tubal orifice was examined from the left side and vice versa to avoid perspective distortion.

For the examination of the nasopharyngeal vault, the posterior wall and the lateral pharyngeal recesses the postnasal mirror was used. In the great majority of the cases a small mirror could be used without any special preparation, but whenever such a technic did not reveal the areas looked for well enough, the uvula retractor was resorted to. Pontocaine, 2 per cent, was sprayed into the pharynx and applied to the posterior surface of the soft palate for a minute

or so, and a self-retaining retractor of the Haslinger type was inserted. A fairly large laryngeal mirror could then be used leisurely and a splendid view obtained. The youngest patient thus examined was about 12 years old. In younger children the retractor was used under general anesthesia. Digital examination was avoided: first, because such a procedure is rather brutal and is likely to render repeated examinations difficult; second, because it was felt that only gross enlargement or absence of the pharyngeal tonsil could thus be ascertained.

The teeth were examined with reference to dental work, extractions, replacements, malocclusion and erupting molars. Dental X-rays were frequently asked for.

The faucial regions were examined with special regard to the condition of the tonsils. If these had been removed, remnants were looked for and the condition of the soft palate noted.

Finally, the posterior pharyngeal wall was carefully checked for the color and moisture of the mucosa as well as for lymphoid follicles and lateral bands.

Under the heading "impression" the examiner attempted to summarize the results of the entire examination as described above. He committed himself freely, regardless of the results of the other phases of the study, such as audiometric tests, general or special physical examinations, and particularly the family history.

The audiometric studies are in the expert hands of Ruth P. Guilder, of the Winthrop Foundation study group. Since the technic of Guilder's methods as well as the interpretation of her observations merit a separate report, the writer shall limit himself here to a brief enumeration and description of the tests used and the means employed. The methods were carefully adapted to suit the age levels. Down to the age of about 3½ years the following tests were applied:

1) Pure tone audiometer for both air and bone conduction.

2) Pure tone audiometer with phonograph attachment for testing with words. This combination was used in two ways: (a) without amplification, to determine the percentage of words actually understood by the patient; (b) with amplification sufficient to raise the score to well over 90 percent. This has proved to be an excellent check on the pure tone audiograms.

3) Weber test with fork of 256 or 512 cycles per second.

4) Rinne test. As a rule a 512 cycles fork with a good AC BC ratio was used.

5) Gelle test was, for purely technical reasons, done by the writer during the clinical examination.

The prekindergarten group, i.e. children under 3½ years of age, comprised only a small percentage of the total number of patients examined so far. The "play-conditioning" technic employed in these cases had heretofore been used only under special school and laboratory conditions. Its use in a clinical setting marks, therefore, a distinct departure from previous practise. This method of testing hearing involves, briefly, the conditioning of the little patient to the sound of bells, rattles, whistles, etc. The responses are obtained by having the child activate various toys.

Treatment should hardly be considered in this paper, but inasmuch as many of the findings demanded treatment or further examinations such as X-ray studies, endocrine studies etc., it may be mentioned here. Actual treatments recommended included inflation, tonsillectomy, and X-ray treatment of the pharynx and eustachian tubes. If the results of the clinical examination and the audiometric studies warranted fenestration operation it was recommended after due discussion among all the members of the team.

SUMMARY

This paper constitutes a part report of the results so far obtained by work that is being carried out under the auspices of the Winthrop Foundation for the Study of Deafness.

A description is given of the procedures used in the clinical ear, nose and throat examination of hard of hearing patients whose hearing disabilities could not readily be ascribed to obvious ear pathology.

In view of the long-range nature of this study, no attempts have been made to evaluate the findings or to draw conclusions.

412 Beacon St.

LXXI

DIAGNOSIS AND TREATMENT OF LABYRINTHITIS SECONDARY TO TYMPANIC INFECTION*

PHILIP MYSEL, M.D.

BOSTON

It was at Dr. Mosher's suggestion that I reviewed the cases of labyrinthitis from the records of the Massachusetts Eye and Ear Infirmary from 1923 to 1933. This study brought out many weaknesses in the diagnosis and treatment of labyrinthitis as a complication of acute and chronic middle ear suppuration. We have learned much since then. This paper deals with our experience in the diagnosis and treatment of labyrinthitis from 1934 to 1942.

The successful treatment of labyrinthitis depends on an early diagnosis. It would be well, therefore, to analyze carefully the signs denoting disturbance in the end organ. The chief objective sign manifesting labyrinth irritation or disease is the presence of a spontaneous nystagmus; the outstanding subjective complaint is a spontaneous vertigo.

Spontaneous nystagmus is an associated movement of both eyes, jerky in character and having a quick and slow component; it is rotary-horizontal in form—the rotary being the chief movement—and the direction of the nystagmus is named after the quick component. We speak of three degrees of labyrinthine nystagmus. By first degree nystagmus, we mean a nystagmus only seen when the patient looks either to the right or left with slightly less than extreme deviation of the eyes, since the latter produces a physiological nystagmus. Second degree nystagmus is seen when the patient looks both directly forward and to the extreme right or left. By third degree nystagmus, we mean that the examiner sees the nystagmus in all three positions of the eyes, i.e., to the right and left, also when the patient looks directly forward. As the patient looks toward the slow component of the nystagmus, the intensity of the latter diminishes, being greater in the first degree position, less in the second degree, and still less in the third. The slow component is the laby-

*From the Department of Otolaryngology of the Massachusetts Eye and Ear Infirmary.

rinthine pull; the quick phase is the cortical pull. Thus in a patient with a destructive labyrinthitis in the right ear, the nystagmus will be most violent when he looks toward the healthy ear, less in the mid-position, and still less when the eyes are directed to his diseased ear. The nystagmus may be coarse, semicoarse, or fine; slow, semiquick or quick. In examining the patient for spontaneous nystagmus, it will be helpful if the subject wears +20 glasses. The wearing of these spectacles magnifies the eyes and cuts out the fixation nystagmus, which in turn reduces its amplitude. If the patient has infection in a previously healthy labyrinth, the amplitude will be great and the frequency marked. On the other hand, a patient who has had a number of attacks of labyrinthitis may show only a fine, slow nystagmus which diminishes in a few hours or days, even though he has an acute destructive labyrinthitis.

We occasionally see a symptomless destruction of the labyrinth, with complete cessation of the acoustic and vestibular functions at the time of examination. In these cases there is no history of vertigo; either the labyrinthine destruction has taken place very gradually or the patient does not recall any attacks of true aural vertigo. Of great importance is the past vestibular history of periodic dizzy spells with turning sensations (objects turn either to the right or left or the patient feels that he is being turned in either direction). Nausea, vomiting, and disturbances in equilibrium should be noted, as well as any periods when the patient has found it necessary to lie down.

If we examine the patient before his attacks of vertigo, we see no spontaneous nystagmus. The past history is, therefore, important. Frequently the attacks of vertigo occurred long ago, and the patient has forgotten about them, or they may have been mistaken for gastrointestinal upsets. In all cases where one suspects labyrinthitis, one examines for disturbances of cochlear function. If the attacks have been mild, the hearing is usually present, but greatly reduced. In severe inflammation of the end organ or after a number of lesser attacks, the hearing is usually (not always) absent. The good ear should be masked by a noise apparatus, otherwise the hearing test will be faulty. The degree of hearing is unimportant; the main question is whether the patient hears anything at all.

In inflammatory disease of the labyrinth secondary to middle ear infection, cochlear function disappears before vestibular function, the former being the more sensitive of the two inner ear functions. We can be sure in end organ disease that if cochlear function is present, vestibular function will likewise be present. Since it is

easy to test a conscious and cooperative patient for the presence or absence of hearing, it will be unnecessary to carry out the caloric, turning and fistular tests in an acutely ill patient. If the vestibular tests are not contraindicated, they should be done. Of late years, we have given up the caloric and turning tests on patients who have labyrinthitis associated with meningeal irritation or with meningitis, relying on the hearing test and the information obtained from the study of the spinal fluid.

For the past seven years, we have used Lund's[1] classification of labyrinthitis. It is based on clinical findings and is simple and easy to understand:

Chronic destructive labyrinthitis includes those cases in which obvious vestibular symptoms are noted (the rare cases of "symptomless" labyrinthine destruction are included) and also those in which no symptoms of vestibular irritation are to be seen at the time of examination, but in which a complete cessation of the acoustic and vestibular functions occurs.

Circumscribed labyrinthitis includes all cases of circumscribed inflammation of the bony wall of the labyrinth with a fistula through the latter, in which the function of the labyrinth remains. When the function has been totally lost, the case would, after clinical lapse, be transferred to the chronic or acute destructive labyrinthitis group.

Serous labyrinthitis includes cases in which obvious vestibular symptoms are observed, and in which there is strongly diminished or temporarily suspended—but returning—function or partial function of the acoustic or vestibular labyrinth, or of both.

Acute diffuse destructive labyrinthitis includes all cases in which the acoustic and vestibular functions are completely and definitely lost in a very short time, either suddenly or in the course of a few days.

Before undertaking operation on cases of chronic destructive labyrinthitis, a careful medical, neurological examination should be made and the spinal fluid analyzed. Epidural and cerebellar abscess, also an associated petrositis must be kept in mind. The failure to take these complications into account is responsible for some of the poor results in the treatment of chronic destructive labyrinthitis. Generally speaking, in this condition a radical mastoid operation should be done, with the removal of all the disease from the vicinity of the oval and round windows and fistulous tracts. We have found that sulfanilamide powder dusted into the middle ear and the mas-

toid bowl has kept local sepsis down to a minimum and prevented postoperative complications so common before the days of chemotherapy. During the past year, we have put our patients on sulfadiazine (in adequate doses) for the first five postoperative days. We feel that it has lessened the danger of intracranial complications.

Lund[1] in 1936 reported 47 cases of chronic destructive labyrinthitis. In 15 of these cases a labyrinth operation was necessary, because an intracranial complication occurred or because the labyrinth was found to be attacked by osseous disease (cholesteatomatous invasion, labyrinthine necrosis, etc.), when the radical mastoid operation was done.

In our own series of cases (at the Massachusetts Eye and Ear Infirmary from 1934 to 1942), we have had 11 cases of chronic destructive labyrinthitis; four required labyrinthectomies, one of which was complicated by a cerebellar abscess. The following examples, taken from this series, present some of the problems associated with chronic destructive labyrinthitis.

CASE 1.—H., a 17-year-old male, was admitted with an old chronic otitis media, dead labyrinth and complete facial paralysis. A radical mastoid operation and decompression of the facial nerve were done (no labyrinthectomy). The patient was readmitted fifteen months later, because he had had four "bouts" of meningitis during the previous two and one-half months. On entrance he showed no meningeal signs and except for 100 cells in the spinal fluid (meningeal irritation), no localizing signs were noted. There had been no return of facial function; there was marked atrophy of the facial muscles. The acoustic and vestibular apparatus showed no response as on the first admission.

The question arose as to whether the patient had a cerebellar abscess—even in the absence of localizing signs—or whether the "bouts" of meningitis were secondary to his dead labyrinth. X-rays of the mastoid and petrous pyramid showed the previous mastoid defect but no destruction in the pyramid. After five days of observation, the headache returned with vomiting, and stiffness of the neck developed. It was decided to do a petrous resection, since the facial nerve was beyond repair and the labyrinth was dead. All bone pathology was removed; the labyrinth was found to be filled with granulations, and cholesteatoma was present in the internal auditory meatus and in the large cells surrounding the porus acusticus. Spinal fluid was obtained on entering the internal auditory meatus. The posterior fossa from the sigmoid sinus to a point just beyond the anterior margin of the internal auditory meatus was removed, also the corresponding tegmental surface; the dural surfaces showed no pathology. We decided to wait for further developments and if necessary to explore the posterior fossa for cerebellar abscess.

The patient made an uncomplicated recovery and six weeks later had a plastic closure of his postaural wound. It is now several months since operation, and he has had no recurrence of his meningitis. Recently a plastic operation was performed to correct his facial deformity.

CASE 2.—Y., a 24-year-old man, was admitted to the Ear Service with an old chronic otitis media. He gave a history of vertigo. Functional testing showed a dead labyrinth; X-rays showed cholesteatomata; the neurological and spinal fluid examinations were negative. A radical mastoid operation was done. The following day the patient had a little frontal headache and a suggestion of stiff neck, but no nausea, vomiting or fever. A spinal tap showed 4200 cells, mainly polymorphonuclear leucocytes (culture sterile). A labyrinthectomy was performed and the meningitis subsided rapidly.

This case, antedating the days of chemotherapy, shows the latency of an old chronic destructive labyrinthitis (dead labyrinth) and how the trauma of the radical mastoid operation precipitated the meningitis.

CASE 3.—S., a male 56 years old, was admitted to the Ear Service with the diagnosis of chronic destructive labyrinthitis and cerebellar abscess. His chief complaint was frontal headache; other important signs were a coarse, horizontal nystagmus to the diseased ear and vomiting. His cochlear and vestibular apparatus showed no function; spinal fluid examination showed 200 cells (60 per cent lymphocytes) and negative cultures. At operation the labyrinth contained granulations and the cerebellum was explored through Trautman's triangle without obtaining pus. Several days later the patient died suddenly from respiratory paralysis, postmortem showing an elongated flat abscess far anteriorly.

During the past nineteen years there have been at the Massachusetts Eye and Ear Infirmary 22 cases of cerebellar abscess secondary to ear infections; eight, or 36.3 per cent, followed chronic destructive labyrinthitis.

We should like here to comment on the use of sulfonamides in the treatment of brain abscess. The sulfonamides are useless unless the abscess can be drained. One should be careful during the period of observation not to continue the drug too long, otherwise it may, for a time, mask the true neurological picture. Recently we had a patient with a right temporal lobe abscess (no localizing signs) in whom the drug masked the meningeal signs so effectively that rupture into the ventricle occurred without warning.

CASE 4.—S., a male 44 years old, was admitted to the Ear Service with chronic destructive labyrinthitis and a partial peripheral facial paralysis. Functional testing showed that the labyrinth was dead. Two weeks previously, removal of a polyp from the right ear had been followed by vertigo and nausea. On entrance, the patient had no headache, vomiting, vertigo or fever. Reflexes were normal; spinal tap showed 400 cells (meningeal irritation) with 50 per cent polymorphonuclear leucocytes and a negative culture. A radical mastoid operation and labyrinthectomy were followed by prompt return of the spinal fluid to normal and clearing up of the facial paralysis.

We see a number of cases of meningitis precipitated by polyp extractions; the latter may start up a labyrinthitis and meningitis or meningitis directly. It is important to know the viability of the labyrinth before polyp removal, since the polypi may be growing

out from a dead labyrinth, and removal through the canal may be promptly followed by meningitis. If the labyrinth is dead, a careful neurological examination should be made ruling out cerebellar abscess and epidural abscess in relation to the base of the petrous pyramid.

A word of caution should be issued against polypectomy in the presence of an active labyrinthitis. If this is done, one may be fairly certain to precipitate a meningitis. Two such cases have recently been seen by the author.

In the treatment of active labyrinthitis without meningitis all surgery is avoided; the patient is kept quiet with phenobarbital and given adequate fluids (by vein if needed). Local treatment consists of gentle cleansing of the ear with a dry pledget of cotton, followed by the instillation of warm boric-alcohol drops three to four times daily. During this period, the patient should be watched for such symptoms as headache, fever, and stiff neck. Spinal fluid examination should be repeated if any such symptoms appear or if the functions of the labyrinth suddenly become extinguished.

Both Lund[2] and Holmgren[3] have drawn attention to the fact that labyrinthogenous meningitis with residual function in the labyrinth may follow a circumscribed or serous labyrinthitis.

Examination of the cerebrospinal fluid is absolutely necessary in every case of manifest labyrinthitis, even if there are no clinical signs that point to an incipient meningeal infection. The presence of such an infection calls for prompt labyrinthectomy.

In circumscribed labyrinthitis (labyrinthine fistula) the fistula is usually located in the horizontal canal; in rare or unusual cases it may be found in the superior or posterior canals. Cholesteatoma is usually present, and in 50 per cent of our cases the fistula symptom was positive. A radical mastoid operation ordinarily gives good results, provided one operates after the vestibular attack has subsided. It will be helpful to keep the patient in bed a week and to control the middle ear infection before doing the radical mastoid operation. The use of sulfanilamide powder locally and sulfadiazine by mouth for several days following the operation has almost done away with exacerbations resulting in acute serous or acute destructive labyrinthitis and meningitis. In general, operation is advisable in this type of case, however, one may try conservative treatment where only the fistulous ear has good hearing function or where the patient refuses operation. Case 5 is an example.

CASE 5.—C., a 60-year-old female, was seen in 1934 with a bilateral chronic otitis media which she had had since early childhood. She had received very little treatment for the previous five years. Outside of deafness, she had no complaints. Examination showed both canals filled with cholesteatomata and X-ray showed large cholesteatomatous cavities in both mastoids. The patient refused operation. After six to eight weeks of treatment, we succeeded in getting both ears free of all cholesteatomata. On the left side, nature had done a beautiful "radical mastoid operation". There was a circumscribed bed of granulations in the region of the horizontal canal. Pressure here with a cotton probe caused marked vertigo and a third degree nystagmus to the opposite ear, although the patient stated definitely that she had never had any attacks of vertigo. The other ear also showed a large cavity due to destruction of the attic wall. We have had this patient under observation for eight years now, and she has no complaints outside of profound deafness. We can always obtain a fistula symptom by pressure on the above area, yet this patient has never had any attacks of vertigo.

Case 5 is cited as an example of what one can do with conservative treatment. In 90 per cent of the cases of circumscribed labyrinthitis, operation is the method of choice.

In serous labyrinthitis we are dealing with manifest vestibular symptoms; hearing is usually gone or suspended temporarily. When serous labyrinthitis follows acute otitis media, it is apt to be associated with meningitis. Facial paralysis increases the gravity of this condition, since it indicates a severe infection not only in the labyrinth but in the paralabyrinthian structures as well. Of our patients with acute serous labyrinthitis with facial paralysis 30 per cent went on to meningitis.

CASE 6.—A. L., a female, aged 20 years, gave a history of chronic otitis media since early childhood. On admission, she showed vestibular symptoms, vertigo, nausea, vomiting and third degree nystagmus to the healthy ear. She was stony deaf on the affected side and had a complete peripheral facial paralysis. Spinal tap was negative; temperature was 99°; there was no headache or stiff neck, etc. She was kept quiet with small doses of sodium luminal hypodermically and given intravenous fluids. Her aural treatment consisted of gentle dry wiping with a cotton probe, followed by the installation of warm boric-alcohol drops four times daily. She was watched closely for signs of intracranial extension (headache, fever and abnormal reflexes). Within three or four days the vestibular symptoms subsided and the affected ear, which had been stony deaf, began to show a slight return of cochlear function. After a quiescent period of three weeks under observation, the patient underwent a radical mastoid operation, without further complication. The facial paralysis which showed some improvement before operation completely cleared following operation.

In less fortunate cases meningeal signs are noted: slight frontal headache, fever and a rise in the cell count of the spinal fluid. It is well to note that meningitis can occur while the labyrinth is still viable. The old theory that meningitis takes place only with the death of the labyrinth is not true. Both Lund[2] and Holmgren[3]

have reported cases of meningitis following circumscribed and serous labyrinthitis with residual function. The signs of early meningeal irritation call for labyrinthectomy, even in the presence of a viable labyrinth.

It is well, at this point, to define what we mean by meningitis. Mygind[4] defines meningitis as an acute febrile disease, caused directly or indirectly by an acute or chronic middle ear suppuration, exhibiting diffuse brain symptoms and a cerebrospinal fluid with pleocytosis, with or without bacteria.

Stewart[5] reported five deaths from meningitis with sterile spinal fluids; out of four post-mortem examinations, two showed streptococci in cultures made from the pus coating the brain (cultures were not made from the other two). He stated that very virulent organisms may often be difficult to culture; on the other hand, organisms that have a low virulence may take on a vegetative rather than a chemical characteristic and may grow profusely on culture. Several years ago, in reviewing a series of meningitis cases at the Massachusetts Eye and Ear Infirmary, we found a number of rapidly fatal cases in which the spinal fluids showed no growth during life.

Neel[6] has, on the basis of 6,004 spinal fluid examinations, proved with certainty that the cerebrospinal fluid is practically devoid of cells, i.e. it contains none or at most two lymphocytes per cubic millimeter. The presence of polymorphonuclear leucocytes is distinctly abnormal. From this it follows that even the presence of several cells means a pathological cerebrospinal fluid, a stage of meningeal irritation, demanding immediate labyrinthectomy. If a labyrinthectomy is performed in cases with minimal incease of cells (ten or more polymorphonuclear leucocytes), one will not be too late. As Lund[1] has pointed out, the examination of the spinal fluid in many cases is the best indication of the "benignity or malignity" of the labyrinthine disease, even better than the examination of the function of the labyrinth. For the past six years, I have tested Lund's views and agree that the rise in the cell count of the spinal fluid is the first sign of meningitis; it precedes the headache, fever and abnormal reflexes. These latter symptoms are frequently late signs of meningitis. Case 7 illustrates this point.

CASE 7.—F., a 30-year-old woman, was admitted with acute diffuse destructive labyrinthitis as the result of an acute exacerbation of an old chronic otitis media. Her hearing was gone. After five days of observation, the cells in the spinal fluid rose to 50, mainly polymorphonuclear leucocytes. A radical mastoid operation was followed by a flare-up of the meningitis, the cells in the spinal fluid rising to 980. A labyrinthectomy was done the next day and the patient given

chemotherapy. On a few occasions the spinal fluid showed hemolytic streptococci, but after a stormy convalescence, the patient was discharged well. It would have been safer for the patient to have had a labyrinthectomy instead of the radical mastoid operation, when the spinal fluid showed 50 cells. The operation on the middle ear spaces aggravated the meningitis. In the days before sulfonamides, the chances of such a favorable result would have been exceedingly slim.

In acute destructive labyrinthitis we have manifest vestibular symptoms—nausea, vomiting, strong nystagmus to the healthy ear and a loss of cochlear and vestibular functions in a few hours or days. The Vienna school of otology taught that the proper treatment was labyrinthectomy, even though no intracranial signs were present, since a large percentage of patients went on to meningitis with the death of the labyrinth.

In 1936 Lund[1] reported: (1) 19 cases of acute labyrinthine destruction without meningitis, with no labyrinthectomy and without a single death; (2) 60 cases of acute destruction with meningitis, all with labyrinthectomy and with 13 deaths, a mortality of 21.6 per cent. Lund's mortality figures are the lowest with which I am acquainted. In the first group noted above, he avoided operation with the attendant possibility of injury to the facial nerve in all 19 cases.

In a series of 22 cases of acute destruction of the labyrinth from 1934 to 1942 at the Massachusetts Eye and Ear Infirmary, we had seven cases of acute labyrinthine destruction without meningitis, with no labyrinthectomies and no deaths; and 15 cases of acute destruction with meningitis, all with labyrinthectomy and with five deaths, a mortality of 33.3 per cent.

The following case of acute diffuse destructive labyrinthitis demonstrates that nature and chemotherapy sometimes help where the operator does not appreciate the true pathological picture:

CASE 8.—F., a 22-year-old woman, was admitted with a chronic otitis, acute diffuse destructive labyrinthitis and a full-blown meningitis, also a complete facial paralysis. X-rays showed an enormous cholesteatomatous cavity. Operation that night consisted of removing the cortex and scooping out masses of cholesteatome. Chemotherapy was started immediately. Spinal tap showed thousands of cells; all cultures proved to be sterile. After several stormy days, the patient recovered from her meningitis and the facial paralysis cleared completely. The postaural wound, however, continued to be foul, and further X-rays showed destruction in the base of the petrous pyramid. The second operation showed the labyrinth as the seat of advanced osteitic disease. Several large fistulae were found in different parts of the labyrinth capsule. There was also advanced necrosis in the solid angle which extended posteriorly as far as the vestibular aquaduct. The facial nerve was seen throughout its entire course from the geniculate ganglion to the stylomastoid foramen. A labyrinthectomy was performed and all the necrotic

bone in the base of the petrous pyramid removed. The postaural wound was left open and allowed to granulate slowly. About six weeks later plastic closure was done.

This patient was fortunate in doing so well. Labyrinthectomy was indicated at the time of the first operation. Chemotherapy plus nature, which had done a beautiful radical mastoid operation and created large fistulae in the labyrinth which afforded pretty good drainage, contributed in no small way to saving the life of this patient. If the necrotic labyrinth and disease in the base of the petrous pyramid had not been thoroughly removed, the patient might well have been a candidate for cerebellar abscess or reactivation of her meningitis.

Some interesting points in the treatment of labyrinthitis are brought out by Case 9.

CASE 9.—C., a man 43 years old, was admitted to the Ear Service with acute destructive labyrinthitis. This followed the removal of a polyp in a nearby hospital. On the day of admission, the spinal fluid showed no cells, the next day 23, and five days later 210, mainly polymorphonuclear leucocytes. A labyrinthectomy was performed and the patient given chemotherapy. He made a prompt and uneventful recovery. In this patient's past history there had been a number of periodic dizzy spells; this was significant and should have called for X-ray studies, neurological, cochlear and vestibular examinations. The operation indicated here was a radical mastoidectomy and not simply a polypectomy.

The results of prolonged delay in the treatment of labyrinthitis are seen in Case 10.

CASE 10.—F., a 33-year-old man, was admitted to the Ear Service with acute destructive labyrinthitis, right facial paralysis and meningitis. He was stony deaf in the affected ear; spinal fluid showed 5,000 cells, mainly polymorphonuclears. All spinal fluid cultures proved to be sterile. A labyrinthectomy was promptly done and the facial nerve found embedded in a sequestrum consisting of part of the horizontal canal. The sequestrum was removed without producing further damage to the nerve. Later the patient showed a partial recovery from his facial paralysis. This case occurred before the use of the sulfa drugs. One notes in the history that the patient, before entering the hospital, was treated conservatively for months by his physician, despite the fact that he complained of severe earache and dizzy spells. These symptoms are danger signals and call for hospitalization and study.

In the treatment of labyrinthitis, it is well to bear in mind that the labyrinthitis may be only part of the picture. It may be associated with a petrositis, which may be the cause of the labyrinthitis or independent of it. Cases 11 and 12 illustrate this complication.

CASE 11.—R., a 47-year-old man, had had a simple mastoidectomy following an acute otitis media in a nearby hospital. This was followed by labyrinthitis and meningitis. A labyrinthectomy was attempted and later the patient was admitted

to the neurological service in a moribund condition. Cultures from the spinal fluid showed Type 3 pneumococcus. He was given large doses of sulfapyridine and serum and made an astounding recovery. At this time X-rays showed extensive destruction in his petrous pyramid. Three weeks later the patient was shown at the neurological conference as a triumph of chemotherapy. We advised, at this point, labyrinthectomy and extirpation of the disease in the petrous pyramid, but the patient was allowed to go home. Two weeks later he was readmitted to the Massachusetts Eye and Ear Infirmary with a full-blown meningitis and in spite of a labyrinthectomy, drainage of the petrous pyramid and chemotherapy, the patient died three days later. The petrositis was probably secondary to the labyrinthitis and demonstrates the importance of getting rid of all bone disease in patients who have meningitis or who have recently recovered from meningitis. This case also demonstrates the insidious character of the Type 3 pneumococcus; it may be dormant for weeks in the bony tissue and then come to life with dramatic suddenness.

The next case is one in which a labyrinthitis was secondary to petrositis:

CASE 12.—T., a 7-year-old girl, was admitted Feb. 27, 1942, with slight headache, moderate rise in temperature, slight stiffness of neck and 100 cells in the spinal fluid, mainly polymorphonuclear leucocytes. The spinal fluid cultures proved to be sterile. She had had a simple mastoidectomy in 1939 and a revision of this operation two weeks before entrance. There were no labyrinthine symptoms. X-rays of the mastoid and petrous pyramid showed decalcification in the petrous pyramid anterior to the labyrinth along the tegmental surface. The mastoid was revised and the base of the petrous pyramid inspected for a lead, but none was found. The antrum was opened to its full extent, exposing the short process of the incus. Although a radical mastoid operation was considered, it was thought best to wait a few days to see whether the child would respond to treatment without sacrificing the middle ear contents. The patient did well immediately following operation; headache and abnormal reflexes disappeared. Sulfadiazine was discontinued on the third postoperative day on account of leucopenia. The middle ear promptly became dry. The patient was observed for six weeks following the operation and at no time did she show any further meningeal irritation or labyrinth symptoms. X-rays of the petrous pyramid at ten-day intervals showed recalcification of the latter. Seven weeks after operation, the postaural wound was closed secondarily and several days later the patient was discharged. In two weeks, the patient was readmitted with manifest labyrinth symptoms—strong nystagmus to the healthy ear, loss of hearing and meningeal signs (37 cells in the spinal fluid, 36 of which were polymorphonuclear leucocytes; cultures were sterile). X-rays at this time showed that the previous area of decalcification had undergone further changes with loss of the tegmental surface. A radical mastoid operation and drainage of the petrous pyramid were followed by prompt recovery.

DISCUSSION

From a study of these cases, I wish to emphasize the following points: There should be no surgery during the period of active labyrinthitis, unless there are threatened intracranial complications. If these complications appear, it is far better to operate in the stage of meningeal irritation when the meningitis is benign. At this time,

labyrinthectomy is recommended, not the radical mastoid operation alone, as the latter merely adds fuel to the fire. I cannot stress this point too much.

Postponement of operation until there were signs of a full-blown meningitis meant death before the days of chemotherapy. Even with the aid of the sulfonamides, the prognosis will be better if operation is done in the stage of meningeal irritation, before micro-organisms have a chance to enter the subarachnoid spaces. Also there will be less risk of cerebellar abscess, as, not infrequently, the cerebellum becomes infected in the stage of meningeal irritation, even though it does not show its true form until some time later.

If operation is undertaken while the labyrinthitis remains localized, there is a great risk of spreading the infection through the pre-formed pathways to the endocranial structures. Some of our cases illustrate this point well.

If the labyrinthitis subsides without any intracranial complications, one should wait several weeks before proceeding with the radical mastoid operation. It is safer to use the electric burr instead of hammer and gouge to prevent undue trauma. Even with utmost caution, one may precipitate a meningitis. The patient should be watched for any meningeal signs, and if they appear, labyrinthectomy should be done immediately.

We have fewer cases of labyrinthitis now than formerly, due to better observation and treatment of chronic otitis media. The importance of the care of the patient with chronic otitis and labyrinthine symptoms is better appreciated today. Before dangerous signs have developed, he is frequently hospitalized for study. He is given a careful neurological examination; fields and fundi are examined; spinal fluid and blood studies are made; functional testing of the cochlear and vestibular apparatus is done; careful medical check-up is given, and X-ray studies of the mastoid, petrous pyramid and base of the skull are carried out. One must always bear in mind that the patient with labyrinthitis may also have petrositis, deep epidural or cerebellar abscess.

Our mortality in labyrinthitis with intracranial complications for the period 1923 to 1933 was 72.2 per cent and for the period 1934 to 1942 was 33.3 per cent. Our results have shown marked improvement, and I believe that they can be still further improved, if one is constantly aware of the importance of the following: (1) early diagnosis; (2) conservative treatment as long as the patient shows no intracranial signs; (3) prompt labyrinthectomy with

the early signs of meningitis, when the meningitis is still localized; (4) the use of sulfanilamide powder locally in all mastoid surgery, as well as sulfadiazine by mouth for several days following the operation.

483 Beacon St.

BIBLIOGRAPHY

1. Lund, Robert: Indications for the Labyrinth Operation, J. Laryng. & Otol. 51:425 (July), 1936.

2. Lund, Robert: Indications de la labyrinthectomie, etc., Ann. des mals de l'oreille, 1922.

3. Holmgren, Gunnar: Fourth Northern Otolaryngological Congress, Helsingfors, 1923, Acta otolaryng. 6:237, 1923.

4. Mygind, H.: Benign Forms of Otogenic Meningitis, J. Laryng. & Otol. 37:597, 1922.

5. Stewart, J. P.: Acute Purulent Meningitis of Aural Origin, J. Laryng. & Otol. 44:225, 1929.

6. Neel, A. V.: Schw. Arch. f. Neurol. u. Psychiat. 15:70, 1924; 17:1, 1925.

LXXII

LATERAL TRANSTHYROID PHARYNGOTOMY

HENRY BOYLAN ORTON, M.D.

NEWARK, N. J.

It has now been almost fifteen years since I first had the opportunity to watch Wilfred Trotter of London demonstrate the operation of lateral transthyroid pharyngotomy. Upon my return from London, Dr. Mosher very kindly permitted me to make plaster models of this procedure in his laboratory in Boston. These were presented with a description of the operation of lateral transthyroid pharyngotomy before the American Laryngological Association twelve years ago.

Naturally, in these fifteen years, additional information and experience have been acquired, and whatever our hopes of methods more specific, less drastic, may be, I feel that the operation of lateral transthyroid pharyngotomy for malignant disease of the pharynx is the most useful means we have, and it is unfortunate that the procedure is not more frequently used. With the added refinements of the preoperative and postoperative care and proper selection of cases, malignant growths of the epiglottis; aryepiglottic fold, especially toward the arytenoid extremity; and the lateral pharyngeal wall outside and away from the arytenoid lend themselves to satisfactory operative treatment, as exemplified in the following two cases:

CASE 1.—A female, 71 years of age, consulted me on Nov. 30, 1935, for slight soreness on swallowing on the left side of the throat, which had been going on for about two months. This discomfort was noticed more when not eating; it was accompanied by slight hoarseness. On examination, with the laryngeal mirror, I noticed an ulceration on the left lateral wall of the pharynx, on a level with the left arytenoid. A biopsy showed carcinoma, Grade II. A lateral transthyroid pharyngotomy was done. This patient has remained well all these seven years (Fig. 1.)

CASE 2.—A male, aged 55 years, was seen on Aug. 16, 1935, complaining of hoarseness with an abnormal sensation on the right side of the throat. Two months previously his tonsils had been removed for this complaint. Examination revealed that the epiglottis and base of the tongue were involved. A biopsy showed carcinoma, Grade III. A lateral transthyroid pharyngotomy was done on Aug. 22, removing the epiglottis and base of tongue by means of surgical diathermy. The patient lived five years following the operation. (Fig. 2.)

Fig. 1.—Case 1. Seven years following lateral transthyroid pharyngotomy for malignancy of lateral wall of the pharynx.

Fig. 2.—Case 2. Five years following removal of epiglottis and portion of base of tongue by lateral transthyroid pharyngotomy.

Cancer in this region does not progress rapidly, and generally after the first appearances of definite suspicious symptoms, which may have extended over a period of several months, the patient is still operable. This entire region of the laryngopharynx is so easily examined that many cases, as the two above, should be recognized early, but only too frequently other operations are performed, as in Case 2, the removal of tonsils, which naturally did not relieve the condition.

With a growth, however, starting in the pyriform sinus, the prognosis is not so favorable, for here the growth is practically symptomless in the early stages, and leads to early involvement of the lateral wall of the larynx on the one side and the thyroid cartilage on the other (Fig. 3). So, it often happens by the time a diagnosis is made, a very extensive operation is necessary, with uncertain results as to the length of time before recurrence.

The operation of lateral transthyroid pharyngotomy is not necessarily limited to malignant conditions of the laryngopharynx. By this method, we have a means that gives adequate access to this area and permits satisfactory treatment of a large number of growths, as in the following case:

Case 3.—I. M., male, aged 35 years, was seen in October, 1939, for a large tumor involving the right side of the pharynx, extending from the top of the

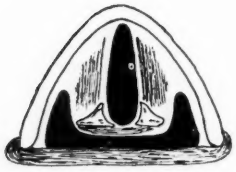


Fig. 3.—Diagram of section through the pyriform sinus of the pharynx to show its relation externally to the thyroid cartilage and internally to the lateral wall of the larynx (Wilfred Trotter).

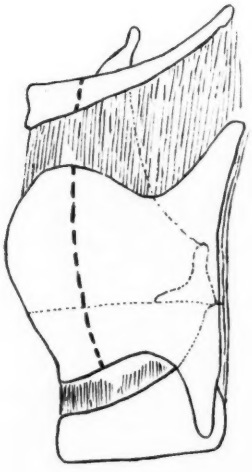


Fig. 4.—The finely dotted lines indicate the relative positions of the aryepiglottic fold, the arytenoid cartilage and the vocal cord. The thickly dotted lines show where the great cornu and the thyroid ala are divided in the operation. When these parts have been removed, the laryngopharynx is accessible. (Wilfred Trotter.)

tonsil in back of the posterior tonsillar pillar, downward so that it pushed the epiglottis upon itself. This was a smooth growth, not painful to the touch. There were no palpable glands in the neck, and the larynx was not involved. This tumor was a neurofibroma, or so-called schwannoma, and was removed by a lateral transthyroid pharyngotomy with no recurrence to date.

The lateral transthyroid pharyngotomy approach is the foundation upon which from the simplest to the most extensive procedure with its reconstruction may be built up. It is a method of access and by removing the great cornu of the hyoid bone and the ala of the thyroid cartilage, the wall of the pharynx is disclosed unopened, palpation can determine with fair degree of precision the extent of growth and the decision as to radical procedure or otherwise (Fig. 4).

A preliminary tracheotomy is necessary, not disturbing the thyroid isthmus, and a disc of trachea is removed. When the glands are involved, a unilateral dissection is done with resection of the jugular vein and ligation of the external carotid; preserving the sterno-mastoid muscle, the anterior edge of which is sutured to the prevertebral fascia behind the pharynx, protecting the great vessels of the neck from infection. This area is drained by a counter-puncture posteriorly.

A vertical incision is then made over the middle of the lateral aspect of the larynx down to the thyroid cartilage, the infrahyoid muscles and constrictor muscles are turned upward and backward, so as to expose completely the hyoid bone and ala of thyroid cartilage. The structures on the deep surface of the hyoid bone and ala of the thyroid cartilage are separated, then the great cornu of hyoid bone is removed by means of heavy scissors, as well as the posterior two-thirds or more of the ala of thyroid cartilage. The pharyngeal wall or aponeurosis is now exposed, and palpation of the growth and extent of the pharyngotomy opening may be ascertained. Having done this, the pharynx is opened, the growth removed (if small) and the gap left is carefully closed and the infrahyoid and constrictor muscles are brought together over the line of incision. In other cases, depending upon the type of procedure, gland resection, in connection with more extensive operations, a plastic procedure will be necessary. (Figs. 5 and 6.)

Generally speaking, with the results of surgery of malignant disease of the laryngopharynx, it is fair to ask what are the results without surgery? There is no more dreadful form of malignant disease and the favorable types left untreated are spared nothing in the end. From an anatomical and surgical standpoint, the operation of lateral transthyroid pharyngotomy gives a splendid exposure

Fig. 5.—Temporary opening in pharynx following an extensive procedure in lateral transthyroid pharyngotomy.

Fig. 6.—Result after first plastic operation to close pharyngeal opening shown in Fig. 5.

to the side of the neck. Lateral transthyroid pharyngotomy in selected cases is not mutilating surgery, but leaves the patient capable of leading a normal useful life.

224 DELAVAN AVE.

BIBLIOGRAPHY

1. Trotter, Wilfred: Purvis Operation on the Surgery of Malignant Disease of the Pharynx, Brit. M. J. 1:269 (Feb. 13), 1926; A Method of Lateral Pharyngotomy for Exposure of Large Growth in the Epilaryngeal Region, J. Rhinol., Otol. & Laryng. 35:289 (Oct.), 1920.

2. Turner, A. Logan: Carcinoma of the Post-Cricoid Region, J. Rhinol., Otol. & Laryng. 35:34 (Feb.), 1920.

3. Orton, H. B.: Lateral Transthyroid Pharyngotomy, Arch. Otolaryng. 12:320-332 (Sept.), 1930.

LXXIII

ORBITAL INFECTIONS*

CHARLES T. PORTER, M.D.

BOSTON

With the rapidity of change in surgical procedures, as well as the introduction of new therapeutic agents, we are forced to re-evaluate our past methods in dealing with all upper respiratory infections. In no department of otolaryngology is this more true than in dealing with infections in the region of the orbit. Here we are dealing with acute infections and the time element is of the greatest importance, and as most orbital infections are of sinus origin a review of the anatomy of the orbit and its relation to the nasal accessory sinuses is in order.

The orbit is a quadrilateral pyramid, base forward. The base is closed by the anterior wall, continuous with the periosteum, the fascia tarso-orbitalis staring from the orbital margin and attached to both tarsi and to the external and internal canthal ligaments. Thus we have a pyramidal shaped cavity whose bony walls, over about one-half of the entire area, are also the walls of the nasal accessory sinuses. In this cavity is the eyeball, optic nerve, extrinsic muscles, ophthalmic artery, the motor nerves, ciliary ganglion and long ciliary nerves; also the superior and inferior ophthalmic veins connecting anteriorly with the veins of the face and sinuses, all of which are without valves and communicate posteriorly with the cavernous sinus. The anterior and posterior ethmoid veins are particularly important. Surrounding the eyeball proper from the cornea to the sheath of the optic nerve is Tenon's capsule. All the interspaces are filled with orbital fat. There are no lymphatic vessels or glands in the orbit.[1] Lying below the orbit is the antrum of Highmore. Mesially are the ethmoid and sphenoid sinuses, while above is the frontal with orbital prolongations and the orbital extension of the ethmoid cells.

We can realize from this that the old procedure, when these cases were rarely referred to a rhinologist and were treated by repeated incision through the lids, is entirely inadequate. These in-

*From the Department of Otolaryngology of the Massachusetts Eye and Ear Infirmary.

fections should be under the combined care of the ophthalmologist and the rhinologist or by one trained in both specialties. Aside from the objective symptoms the patient presents there are several important facts which should be known before we decide upon any course of action. Knowing our anatomy, we must consider routes of infection.

Infections enter the orbit by various pathways, depending on the location of the original source. For example, infections secondary to furuncles, carbuncles and injuries of the face are usually transmitted through the veins of the face. Tooth infections reach the orbit either underneath the periosteum or through the antrum. Infections in the sinuses may enter the orbit by one of several ways: (1) venous thrombosis; (2) thrombophlebitis; (3) direct extension, necrosis of the bone and periosteum; and, of course, any combination of these. In dealing with orbital infection it is well to bear in mind these routes of invasion, for the treatment depends largely upon the source of infection. This is particularly true when we approach the question of differential diagnosis. A careful history, particularly as regards previous upper respiratory infections and sinus involvement and what happened immediately preceding the onset of the infection, such as diving and swimming, is important. Good X-rays are of the greatest value.

After finding out as much as possible in the above manner, we now come to the question of a differential diagnosis. Here the two most serious conditions to be eliminated are osteomyelitis and cavernous sinus thrombosis.

1. In osteomyelitis of the frontal bone the edema may extend downward involving the upper lid and simulating an early orbital infection. One important characteristic of osteomyelitis is the pitting edema extending far up toward the hair line.

It is not infrequent, however, to have osteomyelitis develop from inadequate treatment of orbital infection secondary to acute ethmoiditis. We may have all three conditions present at the same time in the same case. The X-ray and the time element will be of value in diagnosis.

2. In cavernous sinus thrombosis we are apt to depend upon the eyeground changes to help us but they are not infallible. I had one case ending fatally from bilateral cavernous sinus thrombophlebitis with free pus in both sides, with no exophthalmos and normal fundi. The origin of the infection in this case was a chronic ear and mastoid with jugular bulb thrombosis. There was an im-

portant differential diagnostic point, however, in a paralysis of both external recti three days before death. Here temperature does not help us and while the white blood count is usually higher in cavernous sinus thrombosis, this is not always the fact.

3. Other conditions which may confuse us are not so serious and time is usually available in which to work out the diagnosis. In acute dacryocystitis, for instance, there is little or no temperature, low white blood count, localized pain and swelling over the sac region, frequently clear sinuses to X-ray, and a history, usually, of previous epiphora and possibly other similar attacks.

4. In insect bites, bee stings, etc., the history, point of injury, with lack of systemic symptoms, explain themselves.

5. Tooth infections of the upper maxilla may cause in children marked swelling of the cheek and lower lid on the affected side. The temperature and white blood count are seldom elevated. There is no conjunctival change or limitation of motion. The sinuses are clear with possible exception of the antrum on the affected side.

6. History, X-ray and lack of systemic reaction are usually sufficient for diagnosis of tumors, malignant and benign, and mucoceles of the ethmoids and frontals.

7. Another condition to be considered is acute hemorrhage in the orbit from rupture of an aneurysm. Usually too sudden in onset, these cases have severe pain and rapid exophthalmos with possible presence of bruit and absence of X-ray findings in sinuses and negative history.

Infections of the orbit may be divided into three distinct stages: first, edema of the lids; second, edema of the lids with exophthalmos, with or without limitation of motion; third, the same as the second but with evidence of actual abscess formation, pointing in upper or lower lid. Contrary to statements in various texts, I have not found the place of pointing to be indicative of the location of the place of entry of the infection. For example, the infected thrombus from a posterior ethmoid vein may finally point in the outer portion of the upper lid.

In describing the treatment of cases secondary to sinus infection, the method of procedure varies according to the stage or extent of infection present when the patient first comes under observation. Where possible, the patient should be hospitalized, X-rayed and cultured. The cytological and serological examination of the secretions is of no particular importance in these cases. Here we must depend

on two things: whether the culture is one that warrants the use of the sulfa drugs, and whether we should hold off surgical measures directed at obtaining drainage. We believe that all cases should be started on sulfadiazine at once. If the culture shows beta-hemolytic streptococci and progress under sulfadiazine is unsatisfactory a switch to sulfanilamide is indicated. Sulfadiazine should be given in large initial doses, rapidly bringing the blood level up to 10 to 20 mg. per cent and this level maintained for a considerable time. If there is simple edema of the lids with history of recent upper respiratory infection, the conservative or expectant method should be adopted in conjunction with adequate chemotherapy. This includes shrinking of the nasal mucous membrane with pledgets soaked in a 1 per cent cocaine solution, two or three times a day, with warm albolene spray solution, either sprayed or dropped in the nose, in the interim. (I never use adrenalin in an acutely inflamed nose.) Immediately following the shrinking of the mucous membrane, nasal irrigations of warm normal saline should be given and cold compresses should be applied externally.

If after twenty-four to forty-eight hours of this treatment there is no evidence of improvement, or if the condition has progressed, more active measures are necessary. In children, particularly those with cloudy ethmoids and antra, removal of the anterior portion of the middle turbinate and opening of the ethmoid labyrinth, with possible intranasal antrotomy, will frequently be followed by rapid subsidence of symptoms and cure. In an adult it is preferable, depending on the previous history and X-ray findings, to do a radical antrum instead of an intranasal.

If the case presents at first marked edema of the lids, exophthalmos with limitation of motion, radical measures are advisable as soon as a proper blood level of sulfadiazine has been obtained. This means that the infection has already invaded the orbit to the extent of setting up an infiltration of the orbital tissues. I have found the best results from an external ethmoid exenteration with removal of the floor of the frontal, if this is involved, as well as a Caldwell-Luc type of antrum operation. The ethmoid incision should be left open and sulfanilamide powder should be lightly dusted in the wound daily. By this method of attack the orbital periosteal sheath can be palpated and inspected for areas of necrosis, and if none are found most cases will immediately begin to improve so that secondary closure of the wound can be done in a few days with no more subsequent deformity than if the wound were closed at the time of the original operation.

If, through neglect or obstinacy on the part of the patient, nothing is done until there is definite bony destruction in the ethmoids and frontals, as well as actual abscess formation in the orbit, it is necessary to do a modified Killian or an obliterating frontal sinus operation, besides radical surgery on the antra and drainage of the orbit.

In cases due to causes other than sinus infection, the treatment is similar in that it is directed toward removal of the cause. The orbit should not be entered until there is evidence of pointing or the edema and exophthalmos become so marked as to constitute a danger to vision.

We have had too few cases since the sulfa drugs came into prominence to collect a respectable series, but I am convinced that the best results will be obtained by sulfanilamide locally and ample doses of sulfadiazine by mouth. The blood level should be brought to 15 to 20 mg. per cent before operative measures are instituted, or in any case should be started at the earliest possible moment if immediate operation is imperative. Again we are indebted to sulfadiazine for we do not have to wait for a report on the culture before beginning drug treatment. We should not neglect our surgical principles, as is so often done, and depend entirely on these newer methods of therapy as a sure cure, epen if the culture indicates that the drug should work. As we all know from recent work, streptococcus infection plus sulfanilamide does not always equal cure. We have just had a patient in one of our hospitals who entered with an acute orbital infection. The orbit was incised, no attention being paid to the infected ethmoid. When he did not improve sulfanilamide was given. This did not produce the expected result and by the time adequate surgical intervention was undertaken this was a well-developed osteomyelitis. Now, no one can say that the patient would not have had osteomyelitis in any event, but the chances are that if early, adequate surgery had been done along with adequate chemotherapy the outcome might have been different.

In summing up I would say that with adequate chemotherapy plus immune sera, where available, most early cases will subside under conservative methods of treatment. We will have to perform fewer mutilating operations if cases are referred earlier and the number of serious complications will be greatly reduced.

520 COMMONWEALTH AVE.

REFERENCE

1. Fuchs, H. E.: Textbook of Ophthalmology, p. 14, J. B. Lippincott Co.

LXXIV

CAVERNOUS HEMANGIOMA OF THE NASOPHARYNX

JULIO QUEVEDO, M.D.

GUATEMALA CITY

Nasopharyngeal fibromas of the juvenile type occasionally may show such a vascularity that it is difficult to distinguish them from true hemangiomas. Even more interesting, from the histological standpoint, and more rarely seen are fibromas of the nasopharynx with a structure resembling cavernous tissue. However, in these cases, when the tumor has originally been a fibroma, there is always fibrous tissue present, the microscopic diagnosis being definitely established by the finding of the spindle-shaped or star-shaped fibroblasts.

In the case we are reporting here the tumor was formed exclusively of cavernous tissue and there was just enough connective tissue to hold the vessels together. We believe this is a case of true cavernous hemangioma.

On Dec. 17, 1941, a 22-year-old woman was admitted to the Guatemala City General Hospital. Two years previously she started suffering from headaches; at first the pain was mild and generalized in the whole head, but at times it was severe and localized around the forehead. About eighteen months before her admission she noticed slight difficulty in breathing through the right nostril, with a constant sensation of having the nose partially blocked. She then began to suffer from frequent and long colds; each one left, as a rule, a yellowish postnasal discharge, which did not disappear before two or three months. The blockage of the right side of the nose became almost complete and the patient states that the breathing through the right nostril was practically nil by January, 1941. The character of the headache changed; now the patient suffered about twice a week from severe attacks of frontal pain, which lasted from three to eight hours. In July, 1941, she had the first epistaxis; it was from the right side, lasted less than thirty minutes, but was rather profuse.

The woman's voice had obviously assumed a nasal tone and there was also slight difficulty in swallowing: the patient felt "something strange and uncomfortable" in the posterior part of her nose.

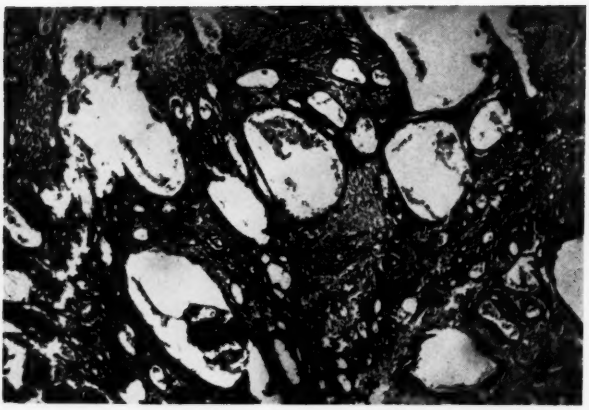


Fig. 1.—This section shows a typical picture of cavernous tissue.

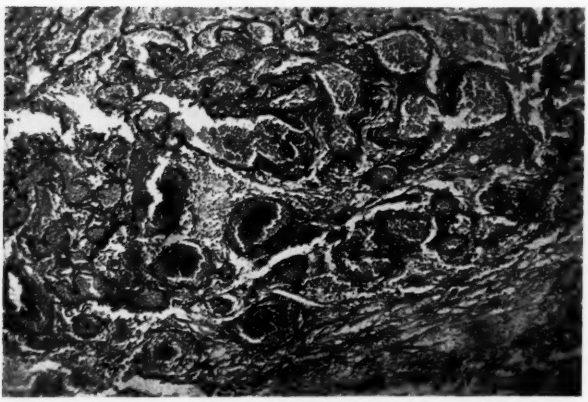


Fig. 2.—In this section the great number of vessels and the scarcity of connective tissue can be appreciated.

Breathing through the left nostril became difficult at times, and there was a continuous colorless discharge from both nasal fossae. On Sept. 15, 1941, there was another hemorrhage, this time from the throat as well as from the right side of the nose; it lasted approximately two hours, but was not particularly severe. Since the onset of the symptoms, this woman had lost twenty pounds. Two months before her admission to the hospital she noticed slight impairment of hearing. Sleeping with the mouth open and snoring were symptoms present almost since the beginning of the disease. Finally, in November, 1941, the patient had profuse bleeding through the throat and through both nostrils, the hemorrhage lasting this time about five hours.

The family history was noncontributory. The patient's own history, outside of the present illness, was limited to measles, whooping cough and intestinal parasites during childhood; mild, easily controlled rheumatism in 1935; occasional fever, attributed to malaria, since 1937. Menstruation started at the age of 14, had always been at regular periods, but had been absent since May, 1941. She had two children.

On admission to the hospital the patient was thoroughly examined and found to have chronic malaria, secondary anemia and a tumor of the nasopharynx. A summary of the blood report was as follows: "4,200,000 red cells; 80 per cent hemoglobin; 9,500 white cells, with 70 per cent polymorphonuclears; positive for Plasmodium malariae; negative Wassermann, Kahn and Jacobsthal reactions." The patient was treated for malaria and anemia, and kept under observation as far as the nasopharyngeal growth was concerned.

We examined the patient for the first time on Feb. 3, 1942. She was a pale thin woman, weighing 105 pounds. According to the records and to the patient's own statement her general condition was very much improved. The only complaints were related to the nasal condition; she had not bled since her admission and the headaches were somewhat relieved. Blood pressure was 110/65.

Local examination showed a normal looking nasal mucous membrane on both sides and the septum slightly deviated to the left. The floor of the nose, on the right side, contained a certain amount of mucopurulent secretion, showing that there was an obstacle in the posterior part of the nose, as it is seen in atresia of the choana or in completely obstructing polypi. The patient could hardly breathe through the right nostril. The left nasal fossa contained a small amount of mucoid secretion and breathing through this side

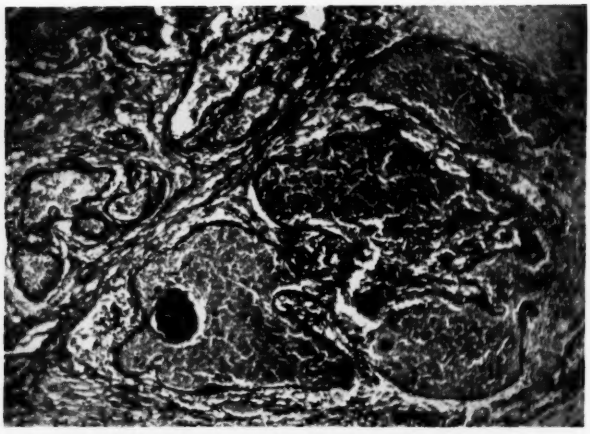


Fig. 3.—This higher magnification shows the thin-walled crowded vessels.

was fair. After cleaning the nose and shrinking the mucous membrane by means of a 1 per cent solution of cocaine, a smooth reddish grey mass could be seen far in the nose, rather into the nasopharynx. There was no appreciable difference between the two sides as far as the tumor mass was concerned. Touching it with a blunt probe, the mass felt elastic, resistant and immobile. Frontal and maxillary sinuses were dull on transillumination. Through the mouth it could be seen that the soft palate had been pushed forward by the nasopharyngeal growth; the hard palate showed no abnormality. The tonsils were small and healthy; the oropharynx, the hypopharynx and the larynx were found to be normal. Posterior rhinoscopy combined with retraction of the soft palate revealed a smooth, moderately lobulated reddish mass filling the nasopharyngeal space. No attachment or adhesions could be seen. Palpation was easily performed and revealed a slightly movable, not very firm mass, about 6 cm. in diameter, attached to the vault of the nasopharynx. Examination with the nasopharyngoscope did not add more information, but confirmed the fact that there was no nasal prolongation of the tumor.

Roentgenograms showed a mass in the nasopharynx; apparently the growth was attached to the vault, but no change in the occipital or sphenoidal bones could be detected. The sphenoidal sinus was

clear and so were the frontals and ethmoidals. The lining of both antra was thickened, but there was no retained secretion.

Examination of the ears revealed marked retraction of both tympanic membranes, and the Rinne test and the audiogram were consistent with conduction deafness.

A provisional diagnosis of nasopharyngeal fibroma was made. Plans for treatment were discussed and surgical removal was decided upon, chiefly because the patient lived far away (Chiapas, Mexico) and could not stay long enough for treatment with radium or electrocoagulation.

On Feb. 6, 1942, the patient was taken to the operating room and a general ether anesthesia, with the patient in the Rose position, was given. The mouth was opened by means of the Davis mouth-gag. Another finger exploration under anesthesia confirmed the previous findings, this time the attachment being definitely established at the uppermost part of the nasopharynx. A rubber-protected snare, with a loose wire, was introduced through the right nostril. With the aid of the forefinger of the left hand the wire loop was passed around the tumor and then the wire was fixed to the snare. Using the short-wave coagulating current the tumor was cut as close to its attachment as possible; after which it was extracted with a tonsil tenaculum. Considerable bleeding ensued and a big postnasal pack had to be inserted. While waiting for the hemorrhage to stop, the tumor was examined and much to our surprise it was found to have shrunk to a small, soft mass measuring about 3 cm. in diameter; it had unquestionably collapsed after removal. The finger did not feel any remaining tumor; the vault of the nasopharynx felt firm and somewhat irregular. The hemorrhage persisted. We thought we were dealing with a cavernous hemangioma and decided to make a curettage of the stump; this was done with a Barnhill adenoid curette. A big postnasal plug was left in place and the patient sent to the ward.

The pathologist, Dr. Carlos Martínez Durán, reported: "The tumor is benign and has all the features of a cavernous hemangioma. The connective tissue is very scarce, hardly enough to separate the big blood lakes."

The postnasal pack was removed at the end of forty-eight hours and there was no more bleeding. The patient made an uneventful recovery and left the hospital on Feb. 20, 1942. Except for the loss of hearing, which remained essentially unchanged, the woman was

relieved of her symptoms. At the time of writing she is still well, and writes that her hearing has improved.

SUMMARY

A case of nasopharyngeal growth is reported. It was diagnosed as fibroma of the juvenile type and was surgically removed. Immediately after removal, macroscopically, and later microscopically, it was found to be a cavernous hemangioma. Photomicrographs are shown.

8 A. S. 46.

LXXV

ENDOSCOPY AT THE CROSSROADS*

LYMAN G. RICHARDS, M.D.

BOSTON

Of the many specialized fields of medicine, few have made more rapid strides than that of otolaryngology. From a position of relative inferiority, with timid surgical foundations and restrictions to conservative office procedures, this specialty has advanced to occupy a distinguished clinical and investigative position which ranks favorably with any other branch of medicine. The exacting demands for surgical skill and anatomical knowledge have been met by an increasing proficiency in the basic and clinical training of the otolaryngologist and the days when a tongue depressor and an atomizer were the chief required armamentarium have receded almost beyond recollection.

In its infancy this specialty was concerned with the area embraced by the ear, nose and visible portion of the throat. Only with the development of the laryngeal mirror by Manuel Garcia in 1858 was it realized that in the larynx lay an intriguing field the study of which laid the foundations of what is properly called laryngology. With training and practice in the use of the mirror and with increasing accuracy and dexterity in indirect manipulation of instruments astonishing proficiency was developed in many intralaryngeal procedures today regarded as outmoded. It was thus quite natural that the more far-seeing and ambitious laryngologists, keenly interested in what lay below the larynx and beyond visibility by means of the laryngeal mirror, should gradually evolve crude but ingenious methods for examination of the tracheobronchial tree and esophagus by direct vision and so take the first halting steps into the field now known as bronchoesophagology. Between 1868 and 1897 a number of pioneers with instruments now worthy only of the museum brought under direct visual inspection much of the upper air and food passages. In 1897 Killian, in many senses the father of bronchoscopy, removed a foreign body from the bronchus and demonstrated the feasibility of lower bronchoscopy. The first authentic

*From the Department of Bronchoscopy of the Massachusetts Eye and Ear Infirmary.

American Bronchoscopy by Coolidge in 1899 effected the removal of a fragment of tracheal cannula from the right bronchus.

Coming on the scene in 1904, Jackson established a new era in bronchoscopy by the perfection and invention of countless ingenious instruments for facilitating the work and in enlarging its scope to include not only the esophagus but the stomach as well. Mosher's brilliant investigations, particularly of the esophagus, added immeasurably to the knowledge of this structure and established him as one of the founders of modern bronchoesophagology.

Stimulated by the work of these pioneers and aided by their ceaseless contributions to instrumental armamentarium and their prolific writing and teaching, more and more men began in various parts of the country to take up bronchoscopy as an expansion of the accepted field of otolaryngology. Almost without exception these men were trained primarily as otolaryngologists and regarded their further progression into the air and food passages as a natural and to a certain extent a deserved right to which their training in monocular vision and their knowledge of laryngeal disease entitled them. Many of these bronchoscopists came to devote a major portion of their efforts to work in the field of pure laryngology and bronchoesophagology and banding together in a common interest founded in 1917 the American Bronchoscopic Society, initial requirements for membership being a fundamental training and experience in laryngology.

In the early days of bronchoscopy essential emphasis was placed on the dextrous removal of foreign bodies from the air and food passages and countless spectacular and life-saving case reports are on record to testify to the skill and courage devoted to this branch of the work. With a steadily increasing number of competent workers and with, it must be confessed, a few bolder but less experienced entrants into this field, the number of foreign bodies falling to the hand of each individual grew necessarily smaller. Excellent postgraduate courses of instruction, invaluable in any individual case, tended nevertheless constantly to enlarge the number of laryngologists who added bronchoscopy to their line of endeavor. There resulted at the same time a progressively growing recognition of the value of bronchoscopic and esophageal examinations, not only for removal of foreign bodies, but as diagnostic procedures in a wide range of pathologic conditions in the upper air and food passages to the point that the highest authorities today place the percentage of foreign body cases at not more than 2 to 3 per cent of the total

number of peroral endoscopic examinations. Moreover it is increasingly evident that therapeutic measures in the air and food passages form an ever-expanding indication for the use of the bronchoscope and esophagoscope and that the internist may profitably turn for help to the endoscopist in much the same manner as to the roentgenologist. Thus from the pioneers' labor of forty years ago has developed a specialized branch of medicine acknowledged and utilized to a degree never dreamed of at its inception.

Until the past five years both diagnostic and therapeutic bronchoesophagology lay almost exclusively in the hands of men trained primarily as otolaryngologists for reasons already explained. Problems in this field were brought to them by internists, thoracic surgeons, and gastroenterologists who sought information by this means which became increasingly valuable to them in their own domain. Gradually, however, the bronchoscopist has begun to sense the slow encroachment on what he had regarded as a traditional preserve by the very confreres to whom he had been accustomed to furnish spcialized information obtained by peroral endoscopy. Thoracic surgeons, expanding their field by increasingly bolder and safer surgery, began either to make their own preoperative bronchoscopic examinations or to have them made by some colleague trained fundamentally not as a laryngologist but as a chest specialist. By the same token certain gastroenterologists began taking up esophagoscopy as an aid in the problems involved in gastroenterology.

The justification for the shift in emphasis away from laryngology as a basic training for bronchoscopy and esophagoscopy is plausible. The thoracic surgeon first of all sees no necessity, so far as his work is concerned, for training in laryngology, provided that the technic of competent bronchoscopy can with practice and patience be mastered by himself or some designated member of his staff. This is also true of physicians who deal largely with pulmonary tuberculosis in which diagnostic and therapeutic bronchoscopy is becoming increasingly feasible and useful. The same applies to a lesser degree to the gastroenterologist who may wish personally to inspect a lesion of the esophagus as an integral part of the gastrointestinal tract. The thoracic surgeon advances the argument that it is of vital importance that he obtain an opinion relative to the location of a bronchial neoplasm or a lung abscess which is backed by a knowledge of pulmonary pathology and surgical anatomy which cannot possibly be expected from the laryngologically trained bronchoscopist whose practical experience in this field is based on the medical school curriculum or at best on a short general internship.

There is no gainsaying the fact that through the medium of excellent postgraduate courses, given incidentally for the most part by men trained in the field of laryngology, the thoracic surgeon, the chest physician or the gastroenterologist can, in a relatively short time, acquire a dependable technic for the safe passage of either the bronchoscope or esophagoscope. In addition, for the gastroenterologist, there is available the flexible gastroscope and peritoneoscope, two amplifications with which the average bronchoscopist of laryngeal training is not ordinarily familiar.

Constantly advanced as another reason for this shifting from one basic medical group to another is that of ready availability. If a surgeon or internist makes his own endoscopic examinations he is never dependent on a laryngological confrere who may reasonably be busy when wanted or who must make his examination in another institution or at a time inconvenient to the surgeon or internist. Particularly is this argument offered in connection with postoperative massive collapse of the lung for which bronchoscopic aspiration is finding a steadily increasing field of usefulness. Indeed so much is prompt bronchoscopic drainage stressed that anesthetists are now being added to those trained in the passage of the bronchoscope in order that almost before a patient has left the operating room, he may to this extent be bronchoscoped and so the delay avoided of sending for some far less available bronchoscopist.

Intratracheal and bronchial instillation of opaque media for lung mapping has for many years been a feature of bronchoscopic diagnosis, implemented largely by the bronchoscopist. Of late the roentgenologist has often included this technicality in his own field, again largely on the grounds of increased facility and availability of personnel.

It is apparent from the above that there is a growing tendency, in many ways for good and sufficient reason, for much diagnostic and therapeutic bronchoscopy to pass from the hands of the laryngologically trained man into those of men for whom this procedure has a special value in their own field, in much the same manner as has cystoscopy for the urologist. The simile is only partial, since the thoracic surgeon, internist and gastorenterologist employ endoscopy only in so far as it applies to their particular domain. Thus removal of foreign bodies continues almost universally to be left to the laryngologically trained bronchoscopist, whereas the urologist employs cystoscopy for whatever condition confronts him, either diagnostically, for the catheterization of the ureters, or operatively for the removal of calculi or in other urological surgery.

It is self-evident that this trend toward a subdivision of the field of peroral endoscopy according to specialized anatomical localities is of vital concern to the laryngologist if he intends to include bronchoesophagology in its broadest aspects as a part of his specialty. To this end he must spend an adequate amount of time in postgraduate study and in specialized courses, he must perfect his operative technic by frequent manikin and laboratory practice and often-times spend a considerable sum on necessary instrumental equipment. Thus prepared to deal with all recognized problems in the domain of bronchoesophagology, barring perhaps the use of the flexible gastroscope and peritoneoscope, is he to find that, aside from the occasional case of foreign body in the air and food passages, the remaining diagnostic and therapeutic work is in the hands of the thoracic surgeon, the internist interested in chest diseases, and the gastroenterologist? The very lack of opportunity to pass the bronchoscope and esophagoscope for diagnostic and therapeutic purposes deprives the laryngologist of most essential and repetitious experience so necessary when the unexpected and possibly difficult problem of a foreign body presents itself. It even becomes a somewhat perplexing problem as to where certain bronchoscopic problems are to find a resting place. Shall the removal of a bronchial or esophageal foreign body be attempted by a laryngologist whose infrequent diagnostic endoscopies may not have lead him into the bronchus for three months or by the medical endoscopist who has perhaps in a week's time performed a dozen bronchoscopies?

This I believe to be a most pertinent question, and one destined to become increasingly important as time goes on. Lest the foregoing reflections and assumptions, based largely on chance conversations with colleagues scattered throughout the country, seem too ill-founded to warrant assertion as facts, I have endeavored, by a poll of 600 prominent thoracic surgeons, chest physicians, gastroenterologists, and laryngologically trained bronchoscopists to obtain an accurate survey of this question and to throw some light on the probable trend which may be expected in the future. The essential purpose of this survey is to clarify the picture for the prospective laryngologist who contemplates the inclusion of bronchoscopy in his field, and so permit him to enter upon his work unhandicapped by any illusions as to the true status of his situation.

BRONCHOSCOPY AND THORACIC SURGERY

To 190 members of the American Association for Thoracic Surgery was sent a simple questionnaire asking for information as follows: 1) Do you do your own bronchoscopies? 2) If not, are they done

by a man trained primarily as a laryngologist? 3) Do you anticipate a continual divorcement of bronchoscopy from the field of otolaryngology?

It is freely acknowledged that such questions cannot always be easily answered by an unqualified "yes" or "no", and that the answer varied many times in accordance with the geographical location of the surgeon and his working facilities. Nevertheless the replies show quite clearly the trend which bronchoscopy is following in the field of thoracic surgery. Questioned as to whether or not they did their own bronchoscopies, 59 (33 per cent) replied in the affirmative, 124 (66 per cent) in the negative. Question 2 was presumably answered only when the surgeon denied doing his own bronchoscopic examinations. In 98 instances (53 per cent) the bronchoscopies were being done by men trained primarily as laryngologists, in 41 (47 per cent), by men not so trained, hence either by general surgeons or internists who had taken up bronchoscopy as a special form of diagnosis applicable to the field of thoracic surgery. Oftimes it appears paradoxically that such bronchoscopic training was given to these men in postgraduate courses presided over by a laryngologist, a truly moving example of the altruistic spirit of medicine.

In several replies the suggestion was advanced that the "surgical" bronchoscopist should best have had some training in laryngology, a combination which at present is not readily available in any practical form. Much more often expressed was the feeling that such bronchoscopic diagnostic work was tending to desert the laryngologist because of his essential unfamiliarity with the many problems facing the thoracic surgeon and of his lack of knowledge of pulmonary pathology. There is no gainsaying the fact that the average laryngologist is far from a qualified expert in diseases of the chest, but opportunities to become such are not easily attainable for most specialists in otolaryngology.

In answering Question 3, there were 117 men (63 per cent) who expressed their belief in the continual divorcement of bronchoscopy from the field of otolaryngology, only 49 (37 per cent) disagreeing with this viewpoint. Numerous answers stressed the chest surgeon's disinterest in removal of any foreign bodies from the lung, but left one to wonder, in the presence of pulmonary suppuration secondary to a foreign body, into whose hands such a problem should rightfully fall.

One prominent thoracic surgeon summed up his opinion as follows: "It is my belief that as more well-trained surgeons enter the field of thoracic surgery it will be found desirable for them to take

on more responsibility entailed in diagnosing and treating endobronchial and esophageal lesions [by bronchoscopy]. At the same time, I believe there will continue to be a certain number of men, not thoracic surgeons [i.e., laryngologists] who will have a definite place in bronchoesophagology."

From these replies certain dependable conclusions may reasonably be drawn. One-third of the thoracic surgeons do their own bronchoscopies. For about one-half of the remaining thoracic surgeons, the bronchoscopic work is done by men not trained as laryngologists. The thoracic surgeons were not queried as to esophagoscopy, but since their surgical field is now more and more embracing the esophagus it is fair to assume that here again a considerable amount of esophageal examination is leaving the territory of the otolaryngologist.

More than two-thirds of these thoracic surgeons predict a continuance of the present trend. They are perhaps biased in their point of view, but the likelihood of further merging at least of the diagnostic work appears unmistakable. Wherever training is given in thoracic surgery, bronchoscopy is more and more routinely being included in the curriculum and such education is bound to bear fruits in the not far distant future.

BRONCHOSCOPY AND THE CHEST PHYSICIANS

Questionnaires were sent to over 80 members of the American Society of Chest Physicians. It was felt that the reaction of this group, not primarily engaged in surgical work, would be an interesting and informative index as to whether diagnostic bronchoscopy were leaving not only the specialized field of laryngology, but even passing from the hands of a surgically to a medically trained physician. With such an expansion bronchoscopy would seem to assume a role somewhat analogous to that of ophthalmoscopy, a diagnostic procedure to be used by a physician in any branch of medicine in which valuable information could with requisite skill be obtained. Admittedly the risk to the fundus from inexpert manipulation of the ophthalmoscope will hardly compare to that of a bungling passage of a bronchoscope. There is however a parallel in the multiplicity of hands into which the bronchoscope is falling.

The questions were the same as those put to the thoracic surgeons. Only 11 (13 per cent) of these physicians claimed to be doing their own bronchoscopies. Of the remainder, 52 (62 per cent) were utilizing the laryngologist while for 27 (32 per cent) the services of someone not trained in laryngology were being obtained.

In spite of this latter situation, however, 56 (67 per cent) of the chest physicians thus interrogated expressed the belief in a continual divorcement of bronchoscopy from the field of otolaryngology. It would seem natural that in this group of men not trained as surgeons, there is found a lessened incidence of personal bronchoscopic work as compared to the thoracic surgeon. Nevertheless, one-third of the work not personally done is referred to men outside the field of laryngology.

A considerable number of men in this group are engaged primarily in the treatment of pulmonary tuberculosis in which bronchoscopy is coming more and more to the fore as a diagnostic and therapeutic measure. Such phthisiologists were almost universal in their advocacy of bronchoscopy by the chest physician. Many felt the value of the opinion of the laryngologist as to conditions involving the larynx but doubted the dependability of the laryngologist's knowledge of pulmonary pathology.

On the whole those men in this group who anticipate a separation of diagnostic bronchoscopy from otolaryngology expressed themselves in favor of this change much more forcibly, and it must be acknowledged, with more enthusiasm than the thoracic surgeon, if colorful comments added to the basic questionnaire are any indication. In common with the surgical group, however, these physicians expressed no leanings toward removal of foreign bodies from the lung, nor, quite naturally toward esophagoscopy, regarding bronchoscopy largely as a valuable aid in their work rather than as a part of the larger field of peroral endoscopy.

ESOPHAGOSCOPY AND GASTROENTEROLOGY

To 117 members of the American Gastroenterological Association went a similar questionnaire varying only in the first question which inquired of each as to whether or not he did his own esophagoscopies. In a negative reply from 112 (96 per cent) it is at once evident that as yet the trend in this direction has scarcely started. Supporting this are the figures in Question 2, indicating that 80 per cent of the gastroenterologist's esophagoscopies are still done by the laryngologist, 20 per cent either by a surgeon or medically trained physician who has specialized in esophagoscopy. Many gastroenterologists acknowledged personal employment of the flexible gastroscope but eschewed esophagoscopy as "too dangerous" and as carrying formidable risk, feeling that information secured by roentgenography is sufficient so far as the esophagus is concerned. One must admit the

high degree of specialization involved in the passage of a rigid esophagoscope by one man and the flexible gastroscope by another.

Despite the situation as evidenced by these figures, 62 per cent of the gastroenterologists expressed the opinion that the future is likely to see the same divorcement of esophagoscopy from otolaryngology as is visualized by the chest physician and surgeons with respect to bronchoscopy. Several gastroenterologists expressed the sincere hope for such a change and believe that esophagoscopy should be in the hands of those interested in the esophagus as primarily a part of the gastrointestinal tract. In one large hospital a curious allocation of work appears in the relegation of esophagoscopies and bronchoscopies to the otolaryngologist, while dilatations for cardiospasm and the use of the gastroscope falls to the gastroenterologist. Again as in the case of bronchoscopy foreign bodies in the esophagus must clearly still seek refuge in the hands of the laryngologically trained esophagoscopist.

BRONCHOSCOPY AN BRONCHOESOPHAGOLOGY

Finally, information was sought from the membership of the American Broncho-Esophagological Society. This group of 63 represents with few exceptions what may be termed the traditional approach to peroral endoscopy via the gateway of laryngology. Practically all of these men began practice as otolaryngologists, adding endoscopy to this field by reason either of special talent or interest and stimulated by the teachings and writings of such pioneers along this path as Killian, Jackson and Mosher. This group have followed the evolution of bronchoscopy from almost purely a means of foreign body removal to its present broader aspects as a valuable diagnostic and therapeutic aid in a wide range of pulmonary and esophageal pathology. In like manner they have expanded the confines of esophagoscopy and in many instances acquired facility with both the rigid and flexible gastroscope. This group can therefore be expected to have very definite and personal reactions to any change in the allocation of peroral endoscopy, regarding which they were sent the following questionnaire. 1) Are you finding an increased tendency for chest surgeons and gastroenterologists to do their own bronchoscopies and esophagoscopies? 2) Do you do endoscopies for thoracic surgeons and gastroenterologists? 3) Do you anticipate a continual divorcement of endoscopy from the field of laryngology? 4) Do you feel that endoscopy should be practiced only by men trained primarily as otolaryngologists?

In answer to Question 1, 60 per cent of the replies were in the affirmative, 40 per cent in the negative. This answer is significant

in that, with the high qualifications entailed in membership in this group, 40 per cent of the work traditionally belonging to them is falling into the hands of the other groups above analyzed for reasons which have already been discussed. Nevertheless, as manifested in the answers to Question 2, there were 84 per cent of the "traditional" bronchoesophagologists who still make endoscopic examinations for chest surgeons and gastroenterologists. This is no more than one would expect in view of the momentum imparted to bronchoesophagology by the customs of the past forty years. It is the trend away from this custom that is our present concern and the object of this investigation. The answers to Question 1 are supported, as might naturally be expected, by those to Question 3 and in almost the same percentage predict a continual divorcement of endoscopy from the field of laryngology. This figure can be taken the more assuredly since it obviously is not open to the charge of wishful thinking but represents an honest opinion however personally distasteful and disappointing.

Replying to Question 4, approximately 60 per cent of the answers express the belief that peroral endoscopy should remain exclusively the preserve of the otolaryngologist while a less conservative remainder are willing to concede that such a background and foundation is not essential for the group here concerned.

A somewhat more detailed analysis and scrutiny of the replies from this latter group is necessary as compared with the replies from the thoracic surgeons, chest physicians and gastroenterologists. The bronchoesophagologists are by the very nature of this situation those most concerned in any trend whereby a specific field of work is, in a perfectly understandable sense, being taken away from them. I shall therefore include a few of the more pertinent observations made by some of the individuals in this group as helpful in illuminating their point of view. Answers to the questions frequently varied with the locality and with its size. "Conditions are different in a smaller town than in a larger city. In the former about 98 per cent of the bronchoscopies and esophagoscopies are for foreign bodies, whereas in the latter, the majority are for diagnostic and therapeutic purposes. Young thoracic surgeons are going to do their own endoscopies as they will want to see the pathology and because the 'average' bronchoscopist is not capable of giving them definite data as to the location of the lesion sought for. The otolaryngologist can only hope to have this work turned to him by becoming really experienced in this phase of bronchoscopy".

This last comment, raises it seems to me the vital point in this controversy, so far as it concerns the otolaryngologist. The few foreign body cases which may come his way will serve but poorly to train him in the very diagnostic and therapeutic procedures which are most essential for him to master if he is to fulfill the qualifications required by the present day thoracic surgeon or gastroenterologist. Seeking this opportunity for more frequent excecutions of diagnostic and therapeutic endoscopy, he continually lacks the practice and training so essential for the occasional but at any given moment, possibly highly difficult foreign body problem. The budding bronchoscopist thus finds himself in a real dilemma and may well ponder the question of how he is to make progress in the face of such a situation. Since six times as many peroral endoscopies are found on the public as on the private wards, the difficulty of meeting the financial hurdle entailed in this problem is too obvious for discussion.

It is an intriguing but not entirely practical suggestion that otolaryngologists should first of all be competent internists. Still more bizarre but thought provoking is the visualization of a new field in which a man will specialize in the anatomical region from the larynx to the chest and pharynx to the stomach, omitting the ear and the nose. In this novel arrangement there would presumably be opportunity to achieve a degree of familiarity with purely pulmonary problems to satisfy the requirements of the thoracic surgeon. Such a radical departure from accepted subdivisions of medical practice certainly still lies in the unpredictable future.

Far from approaching the problem with a selfish desire to exclude newcomers some of the "traditional" bronchoscopists feel that the work should expand in its present direction. As one distinguished endoscopist from the West coast puts it: "Peroral endoscopy does not belong to otolaryngology per se. The fact that otolaryngologists have taken up this work has been an outgrowth of their specialty and most otolaryngologists are not equipped to carry on the work. I have for a long time been attempting to get chest surgeons and physicians interested in endoscopy as for instance, the urologist is interested in cystoscopy. The problems with foreign bodies in the upper air and food passages has led many of us to assume care of diseases of the lungs and lower digestive tract that we did not welcome and in which we were not particularly interested. I am happy that finally the thoracic men are taking up peroral endoscopy. If a man is sufficiently interested, even if an otolaryngologist, in chest diseases and if he can qualify in treating these

conditions, there is no reason why he should not include this field in his work." This broad-minded point of view coincides with that of a thoracic surgeon who says: "It seems to me that there is no valid reason for placing endoscopic work with otolaryngology and it is my impression that the only reason this is done in this country is because of the influence exerted by Chevalier Jackson on this branch of medicine." The debt, however, that bronchoesophagology owes to Jackson whatever its future trends, can never be paid. As Myerson aptly puts it, "He has made a steady succession of brilliant contributions since the early days. He has spread a knowledge of [this] science throughout the world and the present high state of its perfection constitutes a monument to his skill, energy and teaching ability."

Numerous illogicalities are brought to light in such a survey. In one prominent medical center the laryngologists receive their training in bronchoscopy in the Department of Medicine. In another large center bronchoscopy is done both by members of the surgical and the laryngological departments in the same operating room but with quite different equipment.

The ideal solution of the dilemma has apparently been achieved in a few of the largest cities where one man, thoroughly trained, laryngologically and thoracically, in peroral endoscopy has announced himself to his confreres as a "pure endoscopist". As such he has restricted himself entirely to this field, forsaken all other routine otolaryngology and by maintaining adequate, up-to-the-minute equipment in a group of hospitals, he and his associates are able to give twenty-four-hour service whenever required. As a result his otolaryngological brethren have altruistically consigned practically all of their endoscopic work to him, thus avoiding the subdivision of the field among so many aspiring endoscopists that no one of them becomes truly proficient. Moreover this arrangement tends to the solution of the difficulty of availability, a stumbling block for any otolaryngologist busied with the many duties incidental to the rest of his work. Such altruism, however, is not to be found in every large city with the result that in many places there are still found too many endoscopists doing too few endoscopies, to the inevitable proficiency of none of them.

Quite probably this problem does not admit of immediate solution. It is clear from this investigation that there is a strong trend toward endoscopic practice by an increasingly large group of men not trained as otolaryngologists. If the latter are to retain their place in the field so richly planted by the pioneers, it seems clear

that they must somehow prepare themselves more thoroughly in a knowledge of pulmonary and gastroenterological pathology if they are to serve efficiently as the eye-piece of their consultants in diagnostic endoscopic work. The larynx will still continue to require not only specialized knowledge and technical skill for local disease but in it may arise pathology which by extension involves the lower respiratory tract. Such a situation can scarcely receive competent care by the thoracic surgeon or internist. Since practically none of the ascendant endoscopists as one of their number puts it "fool with foreign bodies" (and, it may be conceded, quite wisely), these must still fall to the care of the traditional bronchoscopist. As to how he is to meet the increasing difficulty of maintaining the requisite skill if progressively divorced from all other endoscopic procedures, is, I believe, a question which, though not as yet answerable, must be faced.

319 Longwood Ave.

LXXVI

OBSERVATIONS IN ACUTE OTITIS MEDIA*†

JOHN R. RICHARDSON, M.D.

BOSTON

During the past five years, chemotherapy by the sulfonamide group of drugs has made great strides. The mortality and morbidity of diseases such as pneumonia, peritonitis, septicemia and meningitis have been greatly modified. The results in acute otitis media are less dramatic. Yet otologists believe that due to the use of these drugs the operation of mastoidectomy is far less common than it used to be. The pediatrician and general practitioner are convinced that by giving the proper sulfonamide drug, otitis media may be cured often without the necessity of paracentesis. Accounts of chemotherapy in various popular periodicals cause the laity at times to be critical if the physician does not use one of the sulfonamides. Indeed, when surgical mastoiditis develops in the course of acute otitis media where no chemotherapy has been employed, the otologist is sometimes openly criticized. Many otologists after using chemotherapy and achieving both success and failure are still not sure of their ground.

From personal experience and from reports in the literature a certain amount of bewilderment results. I shall therefore quote some of the representative literature regarding the treatment of acute otitis media by chemotherapy. Second, I shall present statistics obtained in the past five years at the Massachusetts Eye and Ear Infirmary. These statistics relate to patients with acute otitis media who did not have the benefits of chemotherapy. It is my hope that an accurate base line may be derived that will permit a better evaluation of the treatment of acute otitis media with or without chemotherapy.

Horan and French[1, 2] from the Royal Navy Hospital in Chatham, England, report a marked decrease in the incidence of mastoidectomy due to chemotherapy. In 1934 to 1937 there were 607 patients with acute otitis media who received no chemotherapy.

---

*From the Department of Otolaryngology of the Massachusetts Eye and Ear Infirmary.

†A summary of this paper was given before the New England Oto-Laryngological Society at the Massachusetts Eye and Ear Infirmary, Feb. 4, 1942.

There developed 138 cases of acute mastoiditis requiring surgery, or 22.7 per cent. From 1937 to 1939, there were 621 patients treated with chemotherapy and 21, or 3.4 per cent, developed a mastoiditis that required operation.

Fisher[3] in June, 1939, observed 183 patients, most of whom were out-patients whose home care was inadequate. In 95 cases of acute otitis media with a paracentesis, not given sulfanilamide, 66 required mastoidectomy; of these four had septicemia, one died, and three recovered following operation. Out of another group of 88 patients who had paracenteses and were given sulfanilamide, only seven required operation. There were no complications. The average duration of the aural discharge was sixty-five days without chemotherapy and twenty-three days with chemotherapy.

Galloway[4] in December, 1939, comments that in 1935 at the Evanston (Ill.) Hospital there were 37 cases of mastoiditis with five deaths. In 1938 there were five mastoidectomies with no deaths. In this community sulfanilamide was commonly given at or before the onset of otitis media. In the same area the Cook County Hospital has shown no marked decrease in the number of mastoidectomies, although the number of deaths has been reduced. At the Cook County Hospital patients previous to admission had received little or no sulfanilamide.

Bowers[5] in a study of 793 cases of acute purulent otitis media found that 194, or 24 per cent, required mastoid operation. Of this group 387 patients were selected, none of whom had had an ear condition for more than two days; 180 were not given chemotherapy and 24, or 13 per cent, came to mastoidectomy; 207 were treated with chemotherapy and in 12, or 5.8 per cent, mastoidectomy was necessary. The author states that this demonstrates a reduction of one-half in the number of mastoid operations due to chemotherapy. And incidentally that in cases treated properly 11 per cent mastoidectomies show a 50 per cent reduction from the large group of 793 cases taken as they came to the hospital, usually with poor home care. The average duration of aural discharge in 113 patients who received a paracentesis in the hospital and chemotherapy was nine days, while in 118 who did not receive chemotherapy the average duration of discharge was seventeen days.

Braun and Bigler[6] treated 425 children with acute otitis media; half were given chemotherapy and half were not. Myringotomy was done on 50 of the controls and 12 cases required mastoidectomy. Only 35 of the sulfanilamide treated group required paracentesis,

and only four came to mastoidectomy. This meant that mastoidectomy was three times as common in the large group that did not receive chemotherapy. The average duration of discharge was 20.43 days in the control group and 11.58 in the sulfanilamide group.

Bilchick and O'Kane[7] used sulfanilamide in moderate doses in an active out-patient clinic. The drug was started as soon as the diagnosis was established and was continued irrespective of the organism. In a control group of 48 cases four mastoid operations were necessary. In the sulfanilamide treated group of 55 cases, nine required mastoid surgery, three of these being simple mastoid revisions. The average duration of discharge was 12.6 days in the control series and 7.0 days in the sulfanilamide treated series.

If we take these six sets of statistics as fairly representative of the voluminous literature, several facts stand out. First there is a general agreement that the duration of aural discharge is decreased by the administration of chemotherapy. The figures vary. It is of course hard to determine any average, for the duration of discharge in acute otitis media varies greatly with the organism, the time of year, etc. Secondly, the majority of the reports indicate a marked decrease in the incidence of mastoid surgery. Again there is a great variance in the figures. The normal or control group varies from 8.37 to 69 per cent requiring a mastoidectomy. This incidence of mastoidectomy is cut down by chemotherapy to a rough average of 5 per cent. It is hard to get good figures in regard to the incidence of mastoidectomy in acute otitis media. The general practitioner finds the incidence low. The otologist, seeing chiefly complications and hospital cases, finds the incidence high. The third point that I should like to make is that the authors agree that undrained pus in the middle ear or mastoid must be treated surgically. Chemotherapy alone does not and should not be expected to heal such cases. The next point is that there is general accord that mastoidectomies are most frequently necessitated in hemolytic streptococcus and pneumococcus ears. Mastoidectomies are likewise needful in the staphylococcus ears but uncommon in the other groups. Lastly there is a vast literature testifying to the efficacy of chemotherapy in the complications of mastoiditis. There can be no question but that the treatment of meningitis and septicemia has been revolutionized by chemotherapy.

To study otitis media and its best treatment, a bacteriological approach was used. At the Massachusetts Eye and Ear Infirmary during the past five years a careful bacteriological study of cases of acute otitis media was correlated with the subsequent course of the

disease. Out-patients were carefully followed by one man. There were five men who did the work and they deserve credit for their efforts: From the first of December to March, 1937, Dr. John Converse; December to March, 1938, Dr. Joshua Drooker; December to March, 1939, Dr. Robert Snow; December to March, 1940, Dr. Julio Quevedo; December to March, 1941, Dr. Frank Brady. To the observations of these doctors I have added a group from my private practice seen in 1939, 1940 and 1941.

By extending the observations over several years, variations in virulence tend to be ruled out. The same months of each year were used, a period when the incidence of otitis media is neither at its highest or its lowest. No attempt was made to select cases. The patients were taken as they came. In order to obtain the best bacteriological results only those cases of acute otitis media which required paracenteses were used. In other words any patient with acute otitis media whose ear ruptured spontaneously, or whose ear had been opened elsewhere, was not included in this study. Likewise excluded were those patients with acute otitis media who had a postaural abscess in the cavity of a previous simple mastoid operation. The treatment of this small group varies from simple incision and drainage to revision of the mastoid. Discharging ears and previous mastoidectomized ears then are not included in this series.

The cases of acute otitis media were treated in the following manner: The usual criteria indicative of the need for paracentesis were present in each case. The canal was filled with a 70 per cent alcohol wick for five minutes. The drum was opened under strict sterile precautions. Cultures were taken and grown in our own laboratory. A small proportion of cultures were grown both aerobically and anaerobically. The difference was not significant. Typing of the pneumococcus was done at the Massachusetts General Bacteriological Laboratory. The results of the bacteriological findings are tabulated in Table I.

A total of 665 ears are recorded. In 150 ears, or 22.5 per cent, there was no growth on the culture. This may be thought to be due to poor bacteriological technic, or it may be that acute otitis media is a virus infection far more often than we realize. The positive cultures should perhaps be regarded as secondary invaders that flourish only after a virus has paved the way. The streptococcus in its various forms, alpha- and beta-hemolytic, and the nonhemolytic, was present in 31.5 per cent of the total number. The pneumococcus was present in 17.1 per cent. The Staphylococcus albus and Staphy-

TABLE I

Summary of Cultures Obtained at Paracentesis of 665 Cases of Acute Otitis Media

Organism	Cultures								
	Converse 37	Drooker 38	Snow 39	Quevedo 40	Brady 41	Richardson 39-41	Total	Total	Per Cent
Alpha Str. haemolyticus	...	9	1	...	12	...	22		
Beta Str. haemolyticus	26	40	62	17	7	17	169		
Nonhemolytic streptococci	5	...	...	7	6	1	19	210	31.6
Pneumococcus	11	16	46	4	11	26	114	114	17.1
Staph. albus	7	2	13	8	30	5	65		
Staph. aureus	2	4	14	13	19	18	70	135	20.3
Influenza	3	5	2	...	...	4	14	14	2.1
B. subtilis	2	...	...	...	1	...	3		
B. diphtheriae	...	...	...	...	...	1	1		
B. diphtheroid bacilli	2	5	2	...	2	4	15		
B. coli	1	...	2	...	...	...	3		
B. proteus	...	1	...	...	...	...	1		
Gram positive bacilli	...	...	...	...	...	3	3		
Gram negative rods	...	...	6	...	5	...	11		
M. catarrhalis	...	...	3	...	...	1	4		
M. tetragenus	...	...	...	...	...	1	1	42	6.3
No growth	41	18	34	5	37	15	150	150	22.6
Total							665	665	

lococcus aureus together made up 20 per cent of the total group. Assorted organisms were recorded in the remaining cases.

These bacteriological findings agree well with the work of others. While many bacteriological studies are recorded in the literature only a few limit themselves to cultures obtained at myringotomy. Page[8] at the Manhattan Eye, Ear, and Throat Hospital in 1934 reported cultures from acute middle ear infections on which myringotomies were performed. This was before the introduction of the sulfon-

TABLE II

Follow-up of Cases Classified According to Culture at Myringotomy

The number and percentage of mastoidectomies are recorded for the various bacteriological groups. The number of chronic discharging ears that developed is also recorded.

	Cultures									
Organism	Converse 37	Drooker 38	Snow 39	Quevedo 40	Brady 41	Richardson 39-41	Total	Mastoids	Per Cent of Total	Chronic Ears
Hem. Strept.	26	49	51	17	19	17	179	17	9.5	3
Non Hem. Strept.	5			6	6	1	18	1		
Pneumococcus	11	16	41	3	9	26	106	4	3.8	
Staph. albus	7	2	10	8	26	5	58	5		
Staph. aureus	2	4	10	13	17	18	64	2	5.7	
Influenza	3	5	2			4	14	2	14.3	1
Sundry	6	6	11		8	10	41			1
No growth	41	18	31	5	34	15	144	2	1.4	1
Total	100	100	156	52	119	96	625	33	5.3	6

amide drugs. During the year 1934 there were 836 paracenteses. Mastoidectomy was performed in 5.1 per cent. Cultures in 30.1 per cent demonstrated the streptococcus. In the present series there are 31.5 per cent. Page found the pneumococcus in 14.2 per cent. This was almost entirely Type III. We found pneumococcus in 17.1 per cent. For the staphylococcus, both albus and aureus, Page found an incidence of 28 per cent. Our incidence is 20 per cent. Page's percentage of "no growth", 23.3 per cent, agrees closely with our 22 per cent. Page attributes the sterile cultures to faulty technic. The incidence of mastoidectomy in the 1934 series was 5.1 per cent and surgical mastoiditis developed only in the streptococcus, pneumococcus and staphylococcus groups. All of these findings are closely paralleled by the present study. Our incidence of mastoidectomy was 5.3 per cent.

Table II gives the results obtained from following our cases. In the follow-up of patients there is an inevitable shrinkage and the

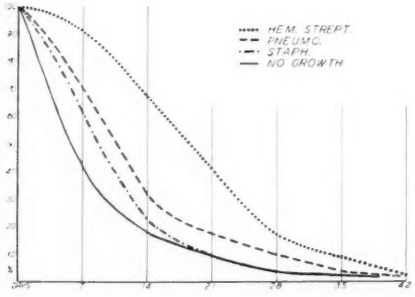


Fig. 1.—A comparison of the rates of healing for the streptococcus, pneumococcus, staphylococcus and "no growth" groups of ears.

total declined to 625. The other cases were lost. The various house officers followed each case until the aural discharge had ceased and the drum had resolved to normal, or the signs and symptoms indicated the need for X-ray. Surgical mastoiditis was operated upon as it developed. The incidence of mastoidectomy in the hemolytic streptococcus group was 9.5 per cent. Of 106 pneumococcus ears only four came to operation, or 3.8 per cent. In the staphylococcus group 5.7 per cent required operation, the larger number being the Staphylococcus albus. Two of the 14 influenza ears came to mastoid operation. None of the ears whose cultures showed the various organisms classified as "sundry" developed surgical mastoiditis. Two of the 114 ears which showed no growth on culture came to operation. The incidence of mastoidectomy in the 625 cases was 5.3 per cent.

Of the six ears listed as chronic discharging, five occurred in the first group of 100 ears treated in 1937. They are described as still discharging, albeit slightly, after two months. I was unable to trace them. The other chronic ear, in the no-growth group, occurred in a 22-year-old man who still had a slight amount of mucopus from his ear seven weeks after paracentesis. He did not return to the clinic. All of the other ears were followed to complete healing either spontaneously or by mastoidectomy.

Let us consider these cases of acute otitis media, grouping them according to the organism cultured at the time of paracentesis. The Streptococcus hemolyticus group was made up of 178 ears. The patients ranged in age from 1 month to 70 years. The average age was a little over 9 years. A bilateral otitis media throughout this study

was classified as two ears rather than as one patient or case. Of this group of 178 ears three became chronic discharging ears as noted above; 17 ears required an operative mastoid exenteration. The average duration of aural discharge of this remaining group of 158 ears was 17.5 days, i.e. 2½ weeks. Fig. 1 shows the time element in weeks plotted against the number of ears. The top curve is plotted from 82 streptococcic hemolytic ears, during the years 1939, 1940 and 1941. The chronic ears are not included. The mastoidectomized ears are not included. Mastoidectomy, it should be repeated, was necessary in 10 per cent of the hemolytic streptococcus ears. Thus the curve shows the rate of healing of 90 per cent of the streptococcus ears. In the first week not many of these ears had dried up and the drums resolved. By the end of the second week almost a third had healed with resolved drums. In the third week a quarter of the cases and in the fourth week another quarter healed. Gradually the curve approaches its base line. The longest duration of aural discharge was 46 days; at the end of five weeks an adenoidectomy was done and in two and one-half weeks more the ear was well. This curve then shows that 36 per cent of hemolytic streptococcus ears do not heal in the first three weeks but do heal in four, five and six weeks without operation.

At any time the signs and symptoms indicating the need for surgery on the mastoid may arise. Surgical mastoiditis occurred in 10 per cent of this group, but is not shown on the curve. Operations were done, one on the fourth day, four on the eighteenth day after paracentesis; one on the nineteenth, the twenty third, twenty-fourth day; two on the twenty-eighth; one on the thirty-fourth, and one on the forty-ninth days. All of these cases operated upon healed uneventfully except one patient who suddenly died of a pulmonary embolus from the leg. This embolus was not related to the ear as shown by autopsy.

Some 20 cases of otitis media included in this group of hemolytic streptococcus ears received sulfanilamide therapy. None of the other 600 cases in the study received chemotherapy unless there were complications after mastoidectomy. Of the 20 treated with chemotherapy, six ears came to mastoidectomy and are included above. The cases operated upon on the thirty-fourth and forty-ninth days after paracentesis are examples. The signs of surgical mastoiditis were masked. Pain occurred; X-rays showed cell destruction which was confirmed by operation. Of the cases receiving sulfanilamide, and healing without complications, the average duration of aural discharge was twenty days. These cases are included in the curve shown

in Fig. 1. The number of cases is so small that it is hardly fair to conclude that sulfanilamide was of no benefit.

Next consider the group where the culture at paracentesis was pneumococcus. There were 106 such ears; 52 of these cultures were typed. Type I pneumococcus was found eight times; Type II in five cultures; Type III in fifteen cultures; Types IV, V, VI one culture each; Type VII seven cultures; Type XIX three cultures; and Type XXIII one culture. The most common types then were I, II, III, VII, VIII, XIV, and XIX.

Of the 106 in this group, 102 ears healed uneventfully. The average duration of discharge was 12.2 days. No chronic discharging ears developed. The second curve again is plotted with time in weeks, against 64 ears that healed in the years 1939, 1940 and 1941. The mastoidectomies (roughly 4 per cent) are not included in the curve. This curve slopes more quickly than the previous one. By the end of the first week 30 per cent of the ears had healed, at the end of the second week two-thirds of the ears were dry with resolved drums, 90 per cent were well at the end of the fourth week. Gradually the curve flattens out. One ear required six weeks to heal and another was not entirely well until nine weeks had elapsed. The graph then demonstrates that the majority of pneumococcic ears are healed within the first three weeks.

Not all ears get well without operation and in the pneumococcus group surgical mastoiditis occurred four times. An ear whose culture showed pneumococcus Type IV required operation on the nineteenth day after paracentesis and healed uneventfully. Three ears with pneumococcus Type III required operation. A 10-month-old child had an antrotomy on the fourth day and healed uneventfully in two weeks. The second of these pneumococcus Type III ears healed in twenty-one days after mastoidectomy. The remaining mastoidectomy, culture showing a Type III pneumococcus, ended fatally. Thirty days after paracentesis mastoidectomy was done and disclosed an extradural abscess. In spite of chemotherapy death occurred two and one-half months after the initial paracentesis from a basilar meningitis. This is a characteristic sequel of Type III infections. Many writers have emphasized the danger of this particular organism. Indeed the incidence of mastoidectomy and of complications is thought by many writers to be much higher than the statistics here presented. Fifteen Type III pneumococcus were found in the half of this group of 106 pneumococcus ears that were typed. There were three Type III mastoids requiring operation in the group of 106.

The group of ears in which cultures at paracentesis showed either the Staphylococcus albus or aureus will next be examined. There were 58 cultures recorded as Staphylococcus albus and 64 as Staphylococcus aureus, making a total of 122. The average duration of discharge was ten days. No ear became chronic although one ear required considerable effort to prevent this. Two adenoidectomies finally resulted in a dry ear six months after the initial paracentesis.

The third curve demonstrates a slightly sharper drop than the previous one. Almost 40 per cent of the ears were healed at the end of seven days; 77 per cent were well at the end of two week, and 90 per cent at the end of three weeks. Like the previous curve, most of the ears were dry at the end of three weeks, the greatest number drying up in the first two weeks.

Five cases of surgical mastoiditis developed in the albus group and two in the aureus group. Therefore the incidence of mastoidectomy is 5.7 per cent for the staphylococcus group; 8.6 per cent for the albus group if it is to be considered alone and 3.1 for the aureus group. There were no complications resulting from operation. A double mastoid operation in a 6-month-old child was slow in healing. One mastoidectomy was done the day following the paracentesis because of postaural swelling. This side healed uneventfully. The other mastoidectomy was performed twenty-eight days after paracentesis. An adenoidectomy was done three weeks later because of persistent discharge from the ear. An operative revision of the mastoid cavity a month later, and four weeks after this, incision and drainage of a cervical abscess were necessary. Eighteen weeks after paracentesis this ear was healed and dry, and the patient discharged as well. Other than this 6-month-old child, the mastoidectomies in the staphylococcus group healed promptly without complications.

The final group of ears are those reported as having "no growth" on culture at the time of paracentesis. There were 144 ears in this category. Pus was obtained at paracentesis in approximately one-third of these. The other two-thirds showed serosanguinous exudate in varying amounts. In about one-third of these ears at myringotomy, the operator noted "gas under pressure". In spite of the "no growth" report one of these ears became a chronic discharging ear; at least it had to be so listed as the patient did not return to the Infirmary after seven weeks. At that time the ear was discharging freely. Two cases showed the signs and symptoms of surgical mastoiditis and were operated upon, both healing uneventfully. The average duration of discharge for 141 ears was 8.8 days.

In Fig. 1 the last curve demonstrates healing in the "no growth" group although only 84 ears are plotted. In the first week, 58 per cent of the ears healed and the drums resolved. By the end of the second week, 82 per cent were healed. The curve gradually flattens down. By the end of two weeks most of the ears were entirely well; more than one-half healed in one week or less.

At the end of the first week 9 per cent of the hemolytic streptococcus ears were healed as compared with 60 per cent of the "no growth" group. At the end of the second week, one-third of the streptococcic group were well and 82 per cent of the "no growth". At the end of the third week 60 per cent of the streptococcic group were well; the same percentage of the "no growth" ears were well in one week. The curves for the pneumococcus and staphylococcus groups fall between.

Remember that all the curves apply to cases not requiring operation for mastoiditis. The curves show how a culture helps in the prognosis. Take the midpoint of the abscissa. One-half of the "no growth" group is well in the first week. One-half of the staphylococcus and pneumococcus groups are well by the end of the second week. One-half of the streptococcic group are not well until the end of the third week.

The culture obtained at paracentesis is also a guide to the incidence of surgical mastoiditis. The incidence of mastoidectomy was approximately 5 per cent for the entire group. When the hemolytic streptococcus was found at paracentesis (31 per cent of the entire group) the incidence of mastoidectomy rises to 10 per cent: one case in every ten. When the culture report at the paracentesis is pneumococcus (17 per cent) 96 cases out of 100 get well. Typing of the pneumococcus is of value, for in the pneumococcus Type III, mastoidectomy is most likely to be necessary and intracranial complications most apt to occur. A staphylococcus report on culture occurred in this series in 20 per cent of the cases. When the staphylococcus is cultured the incidence of mastoidectomy is 5 per cent. There were two mastoidectomies necessary when influenza was cultured. The group is too small to be of statistical value. If the culture report be of any other organism, or "no growth", the chances of mastoid operation are slight indeed.

CONCLUSIONS

From the study of 625 cases of acute otitis media followed from paracentesis to complete healing these observations are made:

1. Culture at myringotomy aids in prognosis and treatment.

2. Culture reports may be expected to show fairly constant relative proportions of the common organisms.

3. The incidence of mastoidectomy varies with the organism found at culture, but for the entire series in this study where chemotherapy was not employed, the incidence of mastoidectomy was only 5.3 per cent.

4. Many cases of acute otitis media that do not cease to discharge in the first two weeks of the disease, heal normally without operation in the third, fourth and fifth weeks.

5. Surgical mastoiditis may develop at any time during the course of otitis media. Operation on the mastoid must then be done in order to cure what may be termed "undrained pus".

6. Using the facts of this study as a base line or norm, certain conclusions in regard to the use of chemotherapy are apparent.

Because of the proved efficacy of the sulfonamide drugs in many diseases surgeons are placing an increased reliance on chemotherapy in acute otitis media. Due to the moral support of this drug therapy, the early or routine mastoid operation done in the first two weeks of the disease, an operation designed to prevent dread complications, has been largely abolished. Today instead of being told "it is safer to operate than to wait" patients are given one of the sulfonamide drugs. This explanation may account for the seeming decrease in the incidence of mastoidectomy reported due to chemotherapy.

As yet chemotherapy, by the sulfonamide group of drugs, is not playing the great role of preventing surgical mastoiditis that it is thought to do by many physicians.

The 5 per cent incidence of cases of acute otitis media requiring a mastoid operation must be materially reduced by chemotherapy in order to justify its routine administration to every case of acute otitis media.

395 COMMONWEALTH AVE.

REFERENCES

1. Horan, V. G., and French, S. Gay: Sulphonamide in Treatment of Acute Mastoiditis, Brit. M. J. 2:942-943 (Nov.), 1938.

2. Horan, V. G., and French, S. Gay: Prevention of Mastoiditis: Surgery of 621 Cases of Acute Otitis Media Treated With Sulfanilamide, Lancet 1:680-682 (April 13), 1940.

3. Fisher, G. E.: Sulfanilamide in Treatment of Otitis Media, J. A. M. A. 112:2271-2272 (June 3), 1939.

4. Galloway, Thomas C.: Chemotherapy, Arch. Otolaryng. 30:901-915 (Dec.), 1939.

5. Bowers, Wesley: Observations on 793 Cases of Acute Purulent Otitis Media With Chemotherapy in 396 Cases, J. A. M. A. 115:178-181 (July 20), 1940.

6. Braun, L. L., and Bigler, J. A.: Sulfanilamide in Treatment of Acute Otitis Media in Children, Am. J. Dis. Child. 62:521-529 (Sept.), 1941.

7. Bilchick, Edwin B., and O'Kane, George H.: Sulfanilamide in Otitis Media in Children, Laryngoscope 51:197-201 (Feb.), 1941.

8. Page, J. R.: Reports of Acute Infections of Middle Ear and Mastoid Processes at the Manhattan Eye, Ear and Throat Hospital During 1934; Their Prevalence and Virulence, Laryngoscope 45:839-843 (Nov.), 1935.

LXXVII

SURGICAL TREATMENT OF OTOSCLEROSIS

COMPARATIVE ANALYSIS OF RESULTS USING DIFFERENT TECHNICS

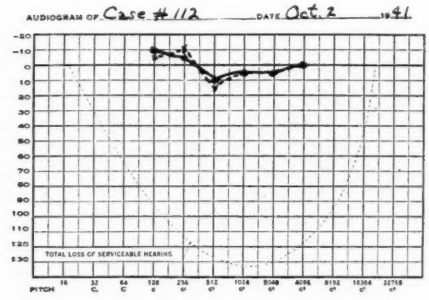
GEORGE E. SHAMBAUGH, JR., M.D.

CHICAGO

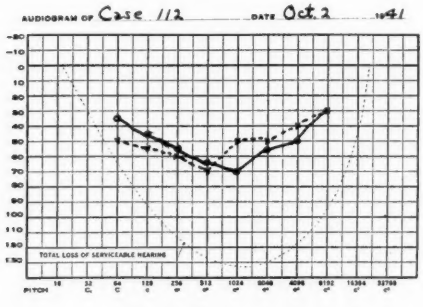
The past several years have witnessed the rapid growth of interest in surgical attempts to improve the hearing in otosclerosis, with the introduction of several different technics or modifications of technic. The value and relative merits of these different technics can be assessed in only one way—by a careful analysis and comparison of the hearing resilts.

Impaired hearing due to otosclerosis is particularly suitable for a comparison of the results of different therapies because of the uniform nature of the pathologic lesion causing the loss of hearing; namely, fixation of the stapes. The technics which have proved successful in improving the hearing surgically all act to correct this obstruction to sound conduction by constructing a new labyrinthine window to take the place of the occluded oval window. Assuming that the stapes fixation is approximately complete, a given technic should restore about the same average number of decibels of hearing in a series of cases. Since variations in the degree of deafness, once stapes fixation has become complete, are due to variations in the amount of secondary nerve degeneration, and since correction of the obstruction to sound conduction by making a new labyrinthine window cannot be expected to improve any of the loss due to nerve degeneration, it is not necessary that the preoperative hearing level be uniform for the results of surgical therapy to be comparable, provided that the diagnosis of approximately complete stapes fixation is correct. Experience has shown that when the hearing by air is 40 to 60 decibels below the hearing by bone the stapes fixation may be regarded as complete or approximately complete, and the cases may be regarded as suitable for operation. (Figs. 1 and 2.)

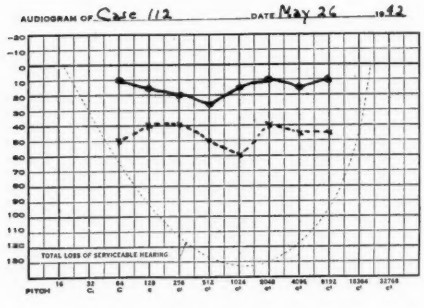
An accurate comparison of results can be made only if sufficient data are available. The minimum data that should be reported are as follows:



A.



B.



C.

Fig. 1.—Ideal case for fenestration operation. This patient, who had worn a hearing aid for six years, has experienced the largest sustained decibel gain for speech (47 decibels eight months after operation) of any patient with otosclerosis thus far reported operated upon by any technic. *A.* Bone conduction before operation. *B.* Air conduction before operation. *C.* Air conduction eight months after operation. Nov-ovalis technic using constant irrigation and the microscope. In these audiograms the dotted line represents the right ear, the solid line the left (operated) ear.

1. Every operated case should be reported.

2. Hearing should be measured audiometrically. A convenient form for recording repeated audiograms is that suggested by Fowler, Jr.,[5] and slightly modified by myself (Fig. 3). The "average for speech" is the average decibel loss for 512, 1024, and 2048. The "change for speech" compares each postoperative speech average with the preoperative average from several preoperative audiograms.

While this method of measuring the hearing for speech fails to take into account the fact that the frequencies of 1024 and 2048 are relatively more important than 512, and the fact that the frequencies of 128, 256, and 4096 are also useful for understanding speech, nevertheless it is relatively accurate as a measure of useful hearing, it is simple and practical, and it has already been used in reports in the literature.

3. There should be adequate controls. The unoperated ear is the best control, and its hearing change after operation should be recorded. An improvement in the control ear as great as in the operated ear cannot be ascribed to the surgical technic.

Because of the normal range of variation in untreated otosclerosis, a change for speech of less than 10 decibels must be regarded as within the limits of variability and as of no consequence.

4. My experience has been that at least six months must elapse before regarding a hearing result after fenestration as probably permanent. Only six months or later should results be considered.

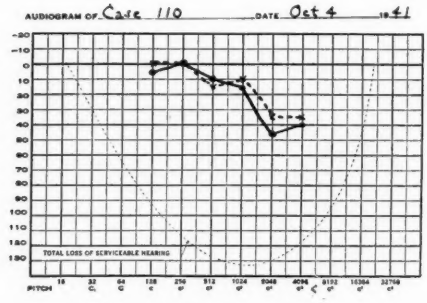
5. All complications and untoward results, including dead labyrinths, should be recorded.

All audiograms should be made under uniform conditions in a quiet inside room and by a person able to make a disinterested and accurate objective test.

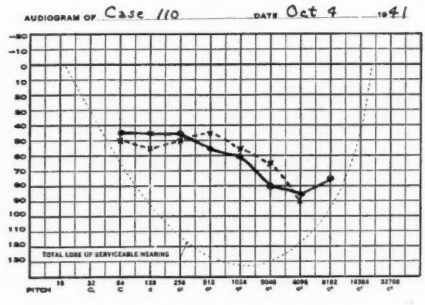
Cases of self-limited chronic hearing loss, such as cases of secretory otitis secondary to eustachian tube occlusion, should of course be excluded by the correct diagnosis of primary stapes ankylosis.

The development and further improvement of the surgical treatment of otosclerosis, and of all other therapy for deafness would be facilitated, and the number of false and misleading reports of therapy would be decreased by complete audiometric reports such as suggested above.

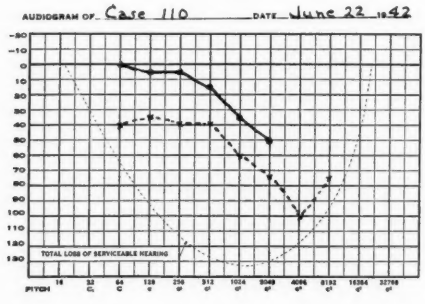
With a few notable exceptions most of the published reports on the surgical treatment of otosclerosis fail to give the complete data required either to evaluate the treatment thoroughly or to com-



A.



B.



C.

Fig. 2.—Case of stapes ankylosis with nerve degeneration suitable for fenestration. This patient can now hear a cat walk across a rug (tones of low pitch) but cannot hear a cat meow (tones of higher pitch). *A.* Bone conduction before operation. *B.* Air conduction before operation. *C.* Air conduction eight months after operation. Nov-ovalis technic using constant irrigation and the microscope.

pare the results with other technics. Such data become increasingly essential to the further development and improvement of the surgical treatment for otosclerosis as new technics and modifications are introduced, for in no other way can their relative merits be judged.

In an attempt to make a more accurate comparison of results with different technics than can be made from the published reports, I have made a comparative analysis of my own results in four series of cases operated upon by different technics. The complete audiometric data with three of these technics are being published elsewhere,[1] and in the interests of brevity will not be duplicated here. These three technics are:

1. The original Lempert fenestration operation (15 ears).

2. The addition to the Lempert technic of a device to provide a continuous flow of normal saline solution across the horizontal canal during the making of the fistula to wash away all blood and bone particles (30 ears).

3. The further addition of a binocular dissecting microscope to facilitate the making of the fistula (66 ears).

In September, 1941, I began to use Lempert's "nov-ovalis" technic modified by the use of constant irrigation and the microscope, and from that date through May, 1942, I operated upon 72 ears by this fourth technic.

Lempert[2] describes the site of the nov-ovalis fenestra as the "dome of the vestibule" and states that the utricle is exposed to view. My observations through the microscope in 85 consecutive nov-ovalis operations have led me to the belief that the utricle itself is not seen, and that the ballooning out of the membranous labyrinth as it runs forward and dips medially represents the ampulla of the membranous canal, the utricle lying immediately medial to it, but out of view. A more accurate anatomical definition of the site of the nov-ovalis fenestra, I believe, would be to call it a fistula into the anterior portion of the bony ampulla of the horizontal semicircular canal. The process of making the nov-ovalis fenestra using constant irrigation and the microscope is illustrated in Fig. 4.

The complete audiometric data on the operations using the nov-ovalis technic modified by irrigation and the microscope are presented in Table II. The only complication has been one case of transient facial paralysis beginning one week after operation and lasting two weeks. There have been no dead labyrinths and no deaths.

SURGEON G.E. Sh. Jr. | Case 63 | DATE & SIDE OF OPERATION Right 1/24/42 Left 3/13/41

Patient's Name Miss N.D. Age 33

Date	RIGHT EAR 64	128	256	512	1024	2048	4096	8192	Average For Speech	Change For Speech	FISTULA TEST (−TO 4+) REMARKS	LEFT EAR 64	128	256	512	1024	2048	4096	8192	Average For Speech	Change For Speech
3/2/41		50	40	40	55	75	55		57				60	50	50	65	80	55		65	
3/8	50	40	45	50	50	70	60	65	57			65	60	55	60	65	75	60	75	67	
3/10	45	40	45	55	55	80	75	65	63			55	50	55	60	70	80	60	80	70	
3/11	45	40	45	55	60	80	65	70	65			55	50	45	55	65	80	80		67	
	Pre-op. level for speech 60											pre-op. level for speech 67									
3/13/41												renestration Left Ear									
3/25	55	50	50	60	65	75	65	70	67	− 7	++++	15	15	20	35	35	60	80		43	+24
4/8	55	40	45	55	60	65	70	70	60	0	"	15	15	15	25	30	35	75		30	+37
4/10	50	45	45	50	60	65	70	70	58	+ 2	"	20	20	10	25	25	30	65		27	+40
4/25	45	45	45	50	55	65	70	70	57	+ 3	"	15	25	15	20	30	30	45	70	27	+40
5/1	45	45	50	55	55	65	65	55	58	+ 2	"	10	15	15	15	25	35	45	65	25	+42
5/26	55	55	55	55	60	65	55	70	60	0	"	25	20	25	25	30	35	40	55	30	+37
10/2	45	50	45	45	50	50	55	50	48	+12	"	20	15	20	20	20	40	55	40	27	+40
12/9	45	45	45	40	55	60	60	60	52	+ 8	"	20	20	20	20	30	35	50	60	28	+39
1/23/42	45	45	45	45	60	70	65	70	58	+ 2	++++	20	20	15	20	30	40	40	60	30	+37
1/26/42	renestration Right Ear																				
2/3	25	20	30	35	40	70	65	70	48	+12	++++	25	20	15	20	35	40	55	60	32	+35
2/12	15	10	15	20	30	55	70	65	35	+25	"	20	20	15	20	35	40	55	65	32	+35
2/21	10	10	15	20	30	45	60	60	31	+29	"	20	20	15	20	30	35	45	70	31	+36
2/28	10	10	10	15	30	40		60	23	+32	"	20	20	15	25	30	40	50	65	31	+36
3/13	15	10	15	20	30	35	55	50	28	+32	"	25	20	15	20	35	40	60	60	31	+36
5/7	15	10	15	20	25	35	60	50	27	+33	"	20	20	15	20	35	40	60	60	31	+36
5/8	10	10	10	15	25	35	60	45	25	+35	++++	20	20	15	20	30	40	50	60	30	+37

Fig. 3.—Form used for recording repeated audiograms, suggested by Fowler, Jr., and modified slightly by the author.

The "probably permanent" hearing results with the four technics in the cases that have been tested six months or more after operation are compared in Table II. With the original Lempert fenestration technic (Technic I) I obtained a hearing improvement of more than 10 decibels for speech six months or more after operation in 27 per cent of the cases. This compares with Campbell's report[3] of a hearing improvement in 35 per cent of 20 cases a year or more after operation. Lempert himself has not presented complete audiometric data on his results of six months or more, but as Fowler, Jr.,[4] pointed out, a comparison of Lempert's first with his later reports indicates that a lasting hearing improvement was secured in 30 per cent of his first ten operated cases.

The addition of constant irrigation to the fenestration technic (Technic II) produced an immediate, striking, and very great improvement in the hearing results so that a hearing gain of more than

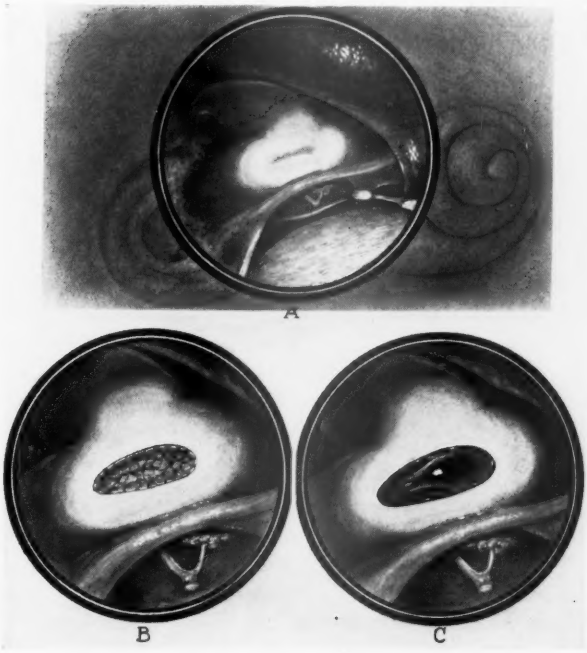


Fig. 4.—Creation of the "nov-ovalis" fenestra using constant irrigation and the binocular dissecting microscope. *A*. Lumen of the bony ampulla of the horizontal canal beginning to appear as a gray streak as seen through the binocular lupe. *B*. Endosteal bone fractured over the bony ampulla as seen through the microscope. *C*. Endosteum removed revealing the ampulla of the membranous canal. Note the nodular otosclerotic bone growing back onto the anterior end of the stapes footplate. (Case 147.)

10 decibels for speech six months or more after operation was secured in 76 per cent of the cases.

The further addition of the use of the microscope to the technic (Technic III) produced a very slight further improvement in results.

The nov-oval's technic modified by constant irrigation and the use of the microscope (Technic IV) seems to be yielding a further slight improvement in the percentage of good results. It must be pointed out that since the nov-ovalis fistula is wider than the fistula in the original Lempert fenestration operation, perhaps bony closure

will take a longer time to become manifest, and perhaps we will find that six months is not long enough to wait before judging the "probably permanent" hearing result in these patients. In this case it may be found that the nov-ovalis technic has no advantage over the original fenestration technic when constant irrigation and the microscope are used.

The comparative analysis of results that I have presented must be regarded in the nature of a preliminary report, presented partly for the information it provides on the comparative merits of different fenestration technics, and partly as a model or example of the complete audiometric data which it is hoped will be provided for each new technic that is introduced, and which are essential if we wish to know the exact hearing results so that we may judge the relative merits of these different technics.

CONCLUSIONS

1. The comparative merits of different surgical technics for improving hearing in otosclerosis can be determined only by a comparison of the hearing results.

2. The minimum data necessary for evaluation and comparison of results are enumerated. All reports of the results of therapy for deafness should present these data.

3. A comparative analysis of the results with four different surgical technics for improving the hearing in otosclerosis shows that:

I. The original Lempert fenestration technic yielded a lasting significant hearing improvement in 27 per cent of cases.

II. The addition of constant irrigation to the technic resulted in a lasting significant hearing improvement in 76 per cent of cases.

III. The further addition of the binocular dissecting microscope to the technic resulted in a lasting hearing gain in 78 per cent of cases.

IV. The nov-ovalis technic with irrigation and the microscope has yielded a hearing gain in 88 per cent of cases that have been tested six months or longer since operation.

122 S. MICHIGAN AVE.

REFERENCES

1. Shambaugh, G. E., Jr.: A Modified Fenestration Technic. Analysis of Results in 127 Consecutive Operations, Arch. Otol. In press.

2. Lempert, Julius: Fenestra Nov-Ovalis: A New Oval Window for the Improvement of Hearing in Cases of Otosclerosis, Arch. Otol. 34:880-912 (Nov.), 1941.

3. Campbell, E. H.: Further Experiences in Fenestration of the Labyrinth in Chronic Progressive Deafness, ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY 50:182-205 (March), 1941.

4. Fowler, E. P., Jr.: Critique of Surgical Treatments for Deafness, Canad. M. A. J. 43:546-552 (Dec.), 1940.

5. Fowler, E. P., Jr.: Personal communication.

TABLE I

SIXTY-THREE EARS OPERATED UPON BY NOV-OVALIS TECHNIC MODIFIED BY USE OF CONSTANT IRRIGATION AND THE DISSECTING MICROSCOPE

Case Number and Ear	Date of Operation	Preoperative Hearing Level for Speech	Best Hearing Result			Final (Most Recent) Hearing Result			
			Time After Operation	Fistula Test	Decibel Change for Speech	Time After Operation	Fistula Test	Decibel Change for Speech	Change for Speech in Unoperated (Control) Ear
104 R	9/22/41	54 db.	2 mo.	++++	+31	2 mo.	++++	+31	+ 9
105 R	9/27/41	52 db.	3 mo.	++++	+39	8 mo.	++++	+29	Operated
106 L	9/29/41	65 db.	3 mo.	++++	+30	9 mo.	++++	+25	— 5
107 R	10/1/41	56 db.	3 mo.	++++	+38	7 mo.	++++	+26	+ 6
108 R	10/2/41	47 db.	2 mo.	±	+ 7	7 mo.	±	0	— 3
31 L	10/4/41	56 db.	6 mo.	++++	+24	6 mo.	++++	+24	Operated
109 R	10/6/41	59 db.	2 mo.	++++	+37	7 mo.	++++	+29	+ 7
11 R	10/9/41	47 db.	2 mo.	++++	+19	8 mo.	++++	+12	Operated
65 R	10/20/41	49 db.	2 mo.	++++	+31	8 mo.	++++	+21	Operated
27 R	10/21/41	60 db.	6 mo.	++++	+35	6 mo.	++++	+30	Operated
110 L	10/26/41	64 db.	2 mo.	++++	+32	6 mo.	++++	+31	Operated
111 R	10/18/41	48 db.	2 mo.	++++	+ 6	7 mo.	++++	0	— 1
13 L	11/3/41	53 db.	1 mo.	++++	+16	7 mo.	++++	+13	Operated
112 L	11/5/41	64 db.	3 mo.	++++	+47	7 mo.	++++	+47	0
113 L	11/10/41	51 db.	2 mo.	++++	+19	7 mo.	++++	+16	—11
114 L	11/13/41	50 db.	6 mo.	++++	+15	6 mo.	++++	+15	— 3
115 L	11/17/41	74 db.	1 mo.	++++	+32	7 mo.	++++	+32	— 4
116 R	11/19/41	56 db.	2 mo.	++++	+26	6 mo.	++++	+21	+ 6
117 R	12/11/41	52 db.	5 mo.	++++	+35	7 mo	++++	+32	— 4
118 R	12/20/41	50 db.	1 mo.	++++	+28	6 mo.	++++	+27	— 2
119 R	12/31/41	53 db.	5 mo.	++++	+30	5 mo.	++++	+30	+ 2
120 L	12/31/41	56 db.	2 mo.	++++	+33	4 mo.	++++	+31	0
121 L	1/2/42	62 db.	4 mo.	++++	+32	5 mo.	++++	+32	+ 2

TABLE 1 (Continued)

Case Number and Ear	Date of Operation	Preoperative Hearing Level for Speech	Best Hearing Result			Final (Most Recent) Hearing Result			
			Time After Operation	Fistula Test	Decibel Change for Speech	Time After Operation	Fistula Test	Decibel Change for Speech	Change for Speech in Unoperated (Control) Ear
122 R	1/5/42	87 db.	2 mo.	++++	+29	6 mo.	++++	+24	Operated
105 L	1/6/42	43 db.	4 mo.	++++	+16	5 mo.	++++	+16	Operated
123 R	1/7/42	52 db.	1 mo.	++++	+31	6 mo.	++++	+29	+ 3
124 R	1/12/42	49 db.	4 mo.	++++	+19	7 mo.	++++	+14	0
125 R	1/13/42	45 db.	2 mo.	++++	+22	7 mo.	++++	+22	+ 5
126 L	1/14/42	64 db.	1 mo.	++++	+31	3 mo.	++++	+29	+ 2
127 R	1/15/42	64 db.	1 mo.	++++	+33	7 mo.	++++	+24	— 3
128 R	1/16/42	54 db.	1 mo.	++++	— 3	6 mo.	++++	—16	— 2
129 R	1/19/42	51 db.	2 mo.	++++	+33	5 mo.	++++	+31	—10
130 R	1/20/42	57 db.	3 mo.	++++	+20	4 mo.	++++	+22	— 1
63 R	1/26/42	60 db.	3 mo.	++++	+35	6 mo.	++++	+33	Operated
131 R	1/27/42	56 db.	4 mo.	++++	+38	6 mo.	++++	+36	+ 4
132 R	1/29/42	69 db.	3 mo.	++++	+11	6 mo.	++++	+ 4	+10
133 L	1/30/42	62 db.	1 mo.	++++	+37	4 mo.	++++	+34	+ 5
134 R	2/2/42	59 db.	4 mo.	++++	+22	6 mo.	++++	+17	0
135 L	2/3/42	64 db.	2 mo.	++++	+31	6 mo.	++++	+29	0
136 R	2/9/42	50 db.	4 mo.	++++	+27	6 mo.	++++	+27	+ 5
137 L	2/10/42	63 db.	2 mo.	++++	+26	2 mo.	++++	+26	+ 1
138 L	2/12/42	52 db.	4 mo.	++++	+29	4 mo.	++++	+29	0
139 L	2/16/42	53 db.	2 mo.	++++	+21	6 mo.	++++	+20	— 6
140 R	2/19/42	54 db.	2 mo.	++++	+27	5 mo.	++++	+27	+ 5
141 R	2/21/42	77 db.	3 mo.	++++	+19	5 mo.	++++	+19	— 6
142 R	2/28/42	53 db.	1 mo.	++++	+23	5 mo.	++++	+16	— 7
44 R	3/5/42	53 db.	3 mo.	++++	+16	3 mo.	++++	+16	Operated
69 L	3/3/42	55 db.	3 mo.	++++	+15	3 mo.	++++	+15	Operated

TABLE I (Continued)

Case Number and Ear	Date of Operation	Preoperative Hearing Level for Speech	Best Hearing Result			Final (Most Recent) Hearing Result			
			Time After Operation	Fistula Test	Decibel Change for Speech	Time After Operation	Fistula Test	Decibel Change for Speech	Change for Speech in Unoperated (Control) Ear
144 L	3/9/42	55 db.	3 mo.	++++	+28	3 mo.	++++	+28	— 3
70 L	3/10/42	65 db.	2 mo.	++++	+17	2 mo.	++++	+17	Operated
145 L	3/13/42	48 db.	1 mo.	++++	+28	5 mo.	++++	+28	+ 3
146 R	3/16/42	66 db.	1 mo.	++++	+36	2 mo.	++++	+34	0
147 R	3/17/42	58 db.	3 mo.	++++	+35	5 mo.	++++	+33	+ 1
148 R	3/19/42	100 db.	2 mo.	++++	+28	2 mo.	++++	+28	
149 R	3/20/42	71 db.	2 mo.	++++	+21	2 mo.	++++	+14	— 2
150 L	3/21/42	53 db.	2 mo.	++++	+26	2 mo.	++++	+26	+ 1
151 R	3/23/42	54 db.	2 mo.	++++	+26	2 mo.	++++	+26	+ 4
153 L	4/23/42	83 db.	3 mo.	++++	+40	3 mo.	++++	+40	+ 4
154 R	4/24/42	70 db.	4 mo.	—	+ 7	4 mo.	—	+ 7	— 5
155 R	4/27/42	65 db.	3 mo.	++++	+18	3 mo.	++++	+18	— 1
156 L	5/1/42	46 db.	3 mo.	++++	+24	3 mo.	++++	+24	+ 2
157 R	5/4/42	69 db.	3 mo.	++++	+36	3 mo.	++++	+36	— 2
158 L	5/5/42	73 db.	2 mo.	++++	+25	2 mo.	++++	+25	— 2
159 R	5/7/42	67 db.	3 mo.	++++	+15	3 mo.	++++	+15	— 5
160 R	5/9/42	61 db.	2 mo.	++++	+14	2 mo.	++++	+14	— 5
161 L	5/11/42	77 db.	2 mo.	++++	+39	2 mo.	++++	+39	+ 7
162 R	5/12/42	49 db.	1 mo.	++++	+31	1 mo.	++++	+31	+16
163 R	5/14/42	60 db.	2 mo.	++++	+28	2 mo.	++++	+28	— 3
164 R	5/15/42	56 db.	2 mo.	++++	+ 3	2 mo.	++++	+ 3	— 3
165 R	5/19/42	50 db.	2 mo.	++++	+23	2 mo.	++++	+23	+ 3
166 R	5/21/42	89 db.	2 mo.	++++	+22	2 mo.	++++	+22	0
167 L	5/26/42	52 db.	2 mo.	±	+29	2 mo.	±	+29	— 3

TABLE II

COMPARISON OF PROBABLY PERMANENT (SIX MONTHS OR LATER) RESULTS USING DIFFERENT TECHNICS

	Number of Ears	Ears Worse (Loss for Speech of 10 db. or More)	Ears Unchanged (Hearing for Speech Within 10 db.)	Ears Improved			Total Showing Gain of More Than 10 db.
				Gain for Speech of 10-19 db.	Gain for Speech of 20-29 db.	Gain for Speech of 30-47 db.	
I. Original Lempert fenestration technic (July 15, 1938, to Jan. 17, 1940)	15	3 (20%)	8 (53%)	4 (27%)	0	0	27%
II. Irrigation added to fenestration technic (Feb. 7, 1940, to Nov. 6, 1940)	29	0	7 (24%)	6 (21%)	10 (34%)	6 (21%)	76%
III. Microscope and irrigation added to technic (Nov. 7, 1940, to Sept. 10, 1941)	55	1 (1.8%)	11 (20%)	17 (30.9%)	16 (29.1%)	10 (18.2%)	78%
IV. Nov-ovalis technic with irrigation and microscope (Sept. 22, 1941, to May 26, 1942)	32	1 (3%)	3 (9%)	6 (19%)	15 (47%)	7 (22%)	88%

LXXVIII

FOURTEEN YEARS OF PRACTICAL EXPERIENCE WITH SINUS PROBLEMS IN AN ALLERGY CLINIC*

FRANCIS L. WEILLE, M.D.

BOSTON

During the past fourteen years, the writer has had the opportunity to observe and treat diseased sinuses in a very large group of asthma cases. A special nose and throat unit has comprised a part of the Allergy Clinic of the Massachusetts General Hospital. Here all special nose and throat problems of allergy clinic patients were evaluated, advice regarding treatment given, and follow-up observation carried out, usually for a long time. Dr. Harris P. Mosher not only originated this research, but constantly gave valuable suggestions and advice in carrying it out.

Seven hundred and eighty-three cases have previously been reported[1, 2] in detail, and a great many additional patients have been studied and followed.

This paper will emphasize certain facts in the light of this experience. The purpose is to clarify the point of view of rhinologists, general practitioners, and allergists in dealing with sinusitis in asthma, especially by expressing some of the fundamental concepts of allergists and rhinologists. Such concepts are not always readily accepted interchangeably by the two types of specialists. The tendency is for each to believe his own therapy the more helpful.

The divergence of opinion regarding the treatment of nasal pathology, especially sinusitis, in asthma, has been due to several factors. Frequently the rhinologist does not understand the underlying problems of allergy and enthusiastically advises some form of nasal surgery, giving a favorable prognosis for both the local condition in the nose and the asthma as well. On the other hand, the allergist has found by experience in following his cases for long periods of time that the enthusiasm of the rhinologist in forecasting a permanent "cure" of asthma by nasal surgery is correct in only one case out of ten.[3, 4] Moreover, he himself is almost equally as

*From the Department of Otolaryngology of the Massachusetts Eye and Ear Infirmary and the Allergy Clinic of the Massachusetts General Hospital.

enthusiastic about some form of therapy for allergy which he hopes will cure or greatly relieve his patient in spite of the fact that only 20 per cent of "cures" are obtained by the combined efforts of allergist and rhinologist. This percentage grows less as the length of follow-up by the allergist himself becomes greater. For example, Rackemann[5] revised his reported series of 213 "cured" cases to 131 after they had been followed for four extra years. It may be remarked that, in general, rhinologists are more apt to report "cures" of asthma from nasal therapy backed up by a short follow-up than are allergists by any form of treatment.

The result of all this is that the allergist is skeptical of the efficacy of nasal treatment for the "cure" of asthma. Conversely, the rhinologist who sees apparent improvement or seeming cure by his own efforts in numerous cases which have had dismal results under the guidance of allergists becomes puzzled over persistence in ineffective therapy on their part and demands that a reasonable time limit be placed on such efforts.

The following points are pertinent in our experience:

1. The allergists have a remarkably good understanding of safe and adequate methods for the parenteral introduction of foreign protein into sensitive patients, and cure probably 20 per cent of their cases of asthma, relieve 60 per cent, and fail in 20 per cent. Yet their understanding of nasal physiology and pathology is inadequate.

2. The rhinologist cures the asthma in about 10 per cent of cases referred to him; relieves for short or long periods (sometimes years) about 40 per cent of such cases; and fails to help the asthma for more than a month (frequently not at all) in 50 per cent of the cases.

Two facts must be emphasized: (a) Such cases include largely the failures of the allergists, and are therefore a special group. Better results might be obtained if there were more cooperation between the rhinologist and the allergist. (b) The above-mentioned figures refer to results with asthma and not results with nasal conditions.

3. The rhinologist by skillful management can obtain varying degrees of improvement in the nasal condition, and usually a satisfactory result (though not cure) in 75 per cent of cases by medical and surgical means combined.

4. It is very rare that an operation on the nose will alone cure the condition in the nose, although it may, from the rhinologist's point

of view, immeasurably lessen the local nasal pathology. (The allergist expects such an operation to cure both the nasal condition and asthma, which almost never happens.)

5. The nasal pathology is probably a part of the asthma, not the cause of it. This is an excellent reason for treating the nose, although it also paradoxically offers an easy excuse for neglect of the nose. Every detail of any disease should be dealt with adequately (not necessarily surgically).

There is good reason to believe that reflex asthma from the nose may be an entity and that postnasal discharge is an important cause for keeping the bronchial portion of broncho-sinus disease going, thereby affecting the bronchioles adversely in asthma. Even with simple thickened membrane in sinuses, gross pus pockets within the membrane itself can be found in one case in nine,[6] thereby raising the "focus of infection" problem in such cases, which include a large group of asthmatics. The problem is greater where purulent or polypoid sinusitis or both are present.

6. As nearly normal nasal physiology as possible must be preserved or restored in asthmatic patients. As a result of the publications of Proetz[8] and others, the very great importance of nasal air-conditioning is being better appreciated by rhinologists who simultaneously are recognizing the fact that self-cleansing of the nose and sinuses by ciliary streaming must not be destroyed by too drastic surgical measures. In other words, the concept of remedying pathological physiology in the nose and sinuses should replace certain former concepts of gross anatomical surgical revisions which sometimes mutilated or destroyed nasal physiological function.

In intrinsic asthma, or infected extrinsic asthma, the tracheobronchial mucosa usually exhibits altered glandular secretion and tissue edema which are important factors in producing wheezing. It is a reasonable hypothesis that physiologically conditioned inspired air is desirable, whereas the contrary is undesirable in these patients.

Because the allergist ordinarily has little knowledge of normal nasal function, the foregoing is not often clearly understood by him.

It should be pointed out that asthmatic patients are often placed in a room which has filtered air and is dust-proof, for both treatment and diagnosis. Normal nasal physiology acts as a dust-filtering mechanism also. Even though this may ordinarily be a means for precipitating asthma by getting the offending allergen in contact with respiratory mucosa in the nose, mouth breathing or inadequate

nasal physiology merely assures a more direct contact of the allergen with lower respiratory tract mucosa.

7. The allergist gets worse results in the nose with his treatment of intrinsic asthma than the rhinologist secures in the asthma by his treatment of the nose!

8. Rackemann[7] has stated that secondary infection makes for failure in the treatment of simple extrinsic asthma. For example, ragweed asthma may continue through the winter if respiratory infection (in the nose or chest) supervenes. Such infection above the carina can be more adequately treated by the rhinolaryngologist than by the allergist, and he should be allowed to try simple medical measures.

9. As the allergist has become more expert in clever detective work which recognizes extrinsic etiological factors, he has accepted the principle that elimination and desensitization are all-important, together with hygiene for improving the general health. If this treatment is adequately carried out, he assumes that a minor burden of nasal infection is harmless and may be forgotten, especially as such therapy is good for the upper respiratory tract. (See numbers 6 and 7 above.)

10. When the allergist is unable to make a diagnosis other than "intrinsic asthma," he usually resorts to vaccine therapy. One benefit of this is said to be due to the foreign protein effect in lessening the severity of acute, subacute, or chronic infection which may be present, or in producing "cure". However, experience has shown that vaccines fail much more frequently if abnormalities are present in the nose and throat.

The fact that asthma has a strong tendency toward remissions may at times explain favorable results from vaccines.

A trial at vaccine therapy should reasonably be limited to a year or less and not prolonged year after year when little or no progress is made.

11. The allergist may be stimulated to have assistance in the management of nasal disease only when the patient becomes desperately ill from asthma, almost as a "last resort". This is too late to be fair to either the patient or the rhinologist.

12. Follow-up observation and treatment, if necessary, of the upper respiratory tract in an asthmatic patient should be coincidental with, in duration, that of the lung pathology. If the one be for years, so should the other.

This is a good argument for making all allergists expert in nose and throat diagnosis and treatment, or vice versa for rhinologists. But better work is done by close cooperation between the two specialties than by combining them, since the rhinologist will, in the average case, do better medical and surgical therapy if he sees a wide variety of pathological conditions besides allergy in his practice, and the average allergist has little time to be a good rhinologist. Moreover, the allergist should rely on direct examination of the nose and throat rather than simple x-ray studies for his information and decisions.

The writer's principles and technic of conservative treatment of sinusitis have been reported recently elsewhere.[4]

CONCLUSIONS

1. This paper emphasizes the need for a clear understanding, and a lack of misunderstanding, of sinusitis in asthma, especially by allergists, rhinologists, and general practitioners.

2. There must be close collaboration between these three types of doctors in the care of such patients.

3. If such collaboration is not possible, it is necessary for the allergist to be a fairly good rhinologist, or for the rhinologist to be well trained in the problems of allergy.

4. Complete self-satisfaction of either the allergist or the rhinologist dealing independently with asthma and with sinusitis in the same patient is probably bad for the patient.

395 COMMONWEALTH AVE.

BIBLIOGRAPHY

1. Weille, Francis L.: Studies in Asthma. XIX. The Nose and Throat in Five Hundred Cases of Asthma, New England J. Med. 215:235-239, 1936.

2. Rackemann, Francis M., and Weille, Francis L.: Nasal Sinusitis and Asthma, Arch. Otolaryng. 30:1051-1060, 1939.

3. Rackemann, Francis M., and Tobey, Harold G.: Studies in Asthma. IV. The Nose and Throat in Asthma, Arch. Otolaryng. 9:612, 1929.

4. Weille, Francis L.: Studies in Asthma. XVIII. The Surgical Treatment of Chronic Sinusitis in Asthma, J. A. M. A. 100:241, 1933.

5. Rackemann, Francis M.: Asthma. XVI. Two Hundred and Thirteen "Cured" Patients Followed Up Four Years Later, Arch. Int. Med. 50:819, 1932.

6. Weille, Francis L.: Asthma. XI. The Pathology of Allergic Tissue as Seen in the Nose and Accessory Sinuses, Arch. Otolaryng. 12:785, 1930.

7. Rackemann, Francis M.: Intrinsic Asthma, J. Allergy 11:147, 1940.

8. Proetz, A. W.: Applied Physiology of the Nose, St. Louis, 1941, Annals Publishing Co.

9. Weille, Francis L.: Treatment of Sinusitis, in press, M. Clin. North America 1942.

The Scientific Papers of the American Broncho-Esophagological Association

LXXIX

ADENOMA (MIXED TUMOR) OF BRONCHUS: A STUDY OF 35 CASES

Louis H. Clerf, M.D.

and

Carl J. Bucher, M.D.

Philadelphia

In a study of bronchial tumors that have come under observation at the Bronchoscopic Clinic of Jefferson Hospital since 1927 there were found 35 that have been diagnosed as adenoma. The true nature of the earlier cases was not recognized and they were considered originally as adenocarcinoma. Their subsequent progress, however, indicated that they did not metastasize, were not influenced by irradiation therapy and could be treated satisfactorily by bronchoscopic removal and by electrocoagulation. Owing to their gland-like structure and apparent benignancy the term "benign glandular tumor of the bronchus" was suggested. These tumors conformed histologically and clinically to the tumors earlier described as endothelioma and adenoma and to the more recently designated "mixed tumor".

Earlier reports would indicate that this type of tumor is rare. During the past fifteen years 243 patients with carcinoma of the bronchus have been diagnosed bronchoscopically at the Clinic. During this same interval 35 patients with adenoma of the bronchus were observed. This constitutes a total of 278 epithelial tumors of which over 12 per cent were adenoma. Obviously this is not an accurate ratio between carcinoma and adenoma since many cases of carcinoma cannot be diagnosed bronchoscopically owing to the location of the tumor. It is apparent, however, that adenoma is not uncommon and must be considered in a study of bronchial tumors. The symptomatology and histopathology of these tumors have been discussed in medical literature and brief reference to these will be

made. The question of therapy, however, still remains a problem and since we have had an opportunity to observe many of these patients over a number of years, emphasis will be placed on this particular part of the problem.

Unlike carcinoma, which occurs more often in the male sex, adenoma is observed frequently among females. In this series of 35 cases there were 19 females (54 per cent) and 16 males (46 per cent).

The age incidence also is of interest. Three of the patients were between 18 and 20 years of age, and 12 were observed between 20 and 30 years, comprising 43 per cent of the group. There were three patients between the ages of 31 and 40 years, 11 between 41 and 50 years, and five between 51 and 60 years. The oldest patient observed was 61 years of age. This is in striking contrast to the age incidence of carcinoma of the bronchus which occurs more often after the fortieth year. It is of interest to note that there were ten females and five males under 30 years of age.

The symptoms were due to bronchial irritation and to obstruction, depending upon the location of the tumor and the degree of obstruction. The marked vascularity of these tumors often resulted in severe hemoptysis.

It was found difficult to elicit from patients information concerning the earliest symptom noted as many of them had been ill for long periods and exhibited advanced pulmonary changes. While cough was the most common symptom, the most alarming was hemoptysis. Cough without or with sputum was observed at some time or other in practically every case. It often was irritative in type and was worse at night particularly when the patient would assume a prone posture. If pulmonary suppuration was present the cough was not unlike that observed in bronchiectasis.

Hemoptysis as an initial symptom was observed in five patients. In two of these the hemorrhages were massive and transfusions were required. In a third the hemoptysis was ascribed to cardiovascular disease and treatment for this was carried out for several years before a roentgen study of the chest revealed a shadow suggesting tumor (Fig. 1). In 16 patients hemorrhage was observed early although it was not the initial symptom, and in an additional nine, blood-tinged sputum was frequently observed. One of the characteristics of the hemoptysis was its sudden occurrence without any premonitory warning. In several patients it occurred at the menstrual period.

Fig. 1.—Roentgenogram of chest of woman, aged 57 years, revealing a sharply circumscribed mass in the middle portion of the left lung. The appearances suggested tumor. There was a history of slight hemoptysis which was considered part of a cardiovascular disorder. Bronchoscopy in 1929 revealed a small tumor projecting from the orifice of the bronchus to the upper lobe of the left lung (Fig. 2). Histologic diagnosis was benign glandular tumor of bronchus. Frequent roentgen studies and bronchoscopic examinations from 1929 to 1934, when the patient died from cardiovascular disease, revealed no apparent increase in the size of the tumor. (Film by Dr. J. T. Farrell, Jr.)

Wheezing respiration as an initial symptom was observed in four patients. In seven additional patients it was noted during the course of the illness. In two patients in whom wheeze was observed as an initial symptom, studies and treatment for an allergic condition had been instituted and only by a roentgen study of the chest was the true cause of the wheeze discovered. It was stated by several patients that the wheeze was influenced by a change in posture. Fever, pain in the chest and dyspnea were not uncommonly noted in patients who had suppuration distal to the obstructing tumor. In one, however, pain in the chest was recorded as an initial symptom.

The physical signs and roentgen findings were dependent upon the degree of obstruction and the extent of the bronchopulmonary changes distal to the growth. Because of the slow growth of this type of tumor extensive bronchopulmonary changes, including pulmonary fibrosis and bronchiectasis, were observed. In 15 patients there was atelectasis of one or more pulmonary lobes. In an additional six there were clinical signs of suppuration with atelectasis. In four

there were evidences of obstructive emphysema. In three the growth could be visualized roentgenologically as a sharply circumscribed tumor mass (Fig. 1). Seven exhibited indefinite findings and clinical and roentgen diagnoses of suppurative bronchitis were made in five instances while a negative report was given in two although there was a definite history of wheezing and occasional blood-tinged sputum. In these two patients there were found small mural growths which produced slight obstruction and no interference of ventilation or drainage of the lung.

From the personal medical history and observations made on patients it would appear that adenoma of the bronchus is a slowly growing tumor (Fig. 2). Many of the patients presented symptoms and signs as well as roentgen evidences of pulmonary diseases for many years (Fig. 3). In addition there were periods of acute pulmonary infection with increased bronchial obstruction and retention of secretions so that diagnoses of pneumonia were not uncommon. In 11 patients the initial diagnosis was pneumonia. In four there was a history of frequent pneumonias. One patient had two attacks of pneumonia yearly for a period of three years. Frequent pleurisy was not uncommon.

In 20 of the 35 patients the symptoms were of more than two years' duration, the longest being eight years. In but seven were the symptoms less than one year in duration. In two of these the first intimation of pulmonary trouble was a copious hemorrhage. A diagnosis of bronchiectasis, based on the instillation of iodized oil, had been made in six cases.

The localization of the growth at the initial bronchoscopy revealed a preponderance of left-sided lesions. In 21 the growth was left-sided, being found in the main bronchus in five cases, at the orifice of the upper lobe bronchus in four and in the lower lobe bronchus in 12. Fourteen cases of right-sided involvement were localized as follows: nine in the lower lobe bronchus, two in the middle lobe bronchial orifice, one in the upper lobe bronchial orifice and two in the main bronchus. No reasonable explanation can be offered for the frequent left-sided involvement.

While the presence of suppuration may influence the endobronchial appearances, the common finding is partial or complete occlusion of a bronchus by a relatively smooth, pinkish, reddish or purplish mass which may at times appear fleshy. The mass may be quite firm or may be soft. As a rule the soft tumor is very vascular and bleeds promptly when touched with an instrument or when a biopsy is

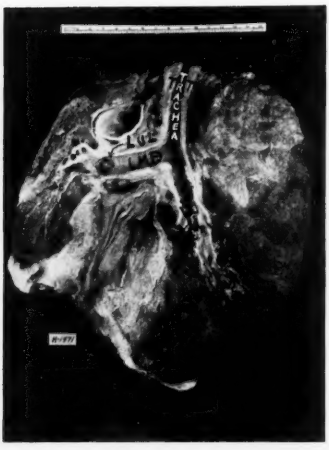


Fig. 2.—Trachea and both bronchi with lungs removed at autopsy in case of adenoma of bronchus (see Fig. 1). Death resulted from cardiovascular disease in 1934. The posterior wall of the trachea (*T*), right main bronchus (*RMB*) and left main bronchus (*LMB*) has been removed and a considerable portion of the left lung and the lower lobar bronchus have been resected to afford a view of the extrabronchial portion of tumor as well as the small mass of growth projecting from the orifice of the upper lobar bronchus (*LUL*). The visible endobronchial portion remained practically unchanged from 1929, when first seen, to 1934.

done. Although usually covered with epithelium, superficial erosions may be present particularly if there is associated suppuration. The smaller tumor may appear as a sessile elevation and suggest that it is endobronchial (Fig. 7*B*). Very commonly, however, the bronchus is filled with a large mass which appears pedunculated and a considerable portion of the tumor may be cored out with the tip of the bronchoscope, or removed with forceps. As a rule there is no fixation or rigidity of the bronchial wall as is seen in carcinoma; if there is associated suppuration and fibrosis the normal flexibility of the bronchus may be interfered with. The pedunculated type of tumor tends to grow toward the trachea so that one may readily move the proximal portion with the tip of the bronchoscope.

Bronchial adenomas can be divided histologically into three fairly distinct groups. The pattern depends upon the type of cell and the arrangement of the gland-like tissue, the amount and character of the interstitial tissue, and the arrangement of blood vessels

and vascular spaces. In a single tumor, there are sometimes transitions from one type of structure to another.

In the first type of adenoma (Fig. 4) the tumor is divided into a number of alveolar spaces by fibrous tissue septa. Within them there are numerous collections of cells arranged in cords or in rosette-like fashion about small open spaces. The cells are of moderate, uniform size and stain about equally in intensity. They are somewhat round, have a moderate amount of acidophilic cytoplasm, and have round and oval vesicular nuclei. Between the cell aggregations, there is a moderate amount of fibrous tissue, and numerous thick- and thin-walled capillaries.

In the second variety of bronchial adenoma (Fig. 5) the septa dividing the tumor into alveoli are present, but they are less apparent. The glandular elements overshadow the other structures. The gland-like cells are abundant and are arranged in pseudo-glandular clusters and cords. Although uniform in size and structure, they appear to be somewhat larger and have a more abundant cytoplasm than those of the first type. The rosette-like arrangement is present, but the open spaces at the center are small or obliterated by the encroachment of the mass of cells upon them. The fibrous tissue separating the pseudo-glands is scant, but the tumor is vascular.

The structure of adenomas of the third type (Fig. 6) differs from the other two in the rather marked tubular character of the pseudo-glands and the large amount and character of the interstitial tissue. The tubular gland-like structures are sometimes solitary but are more often in well-defined clusters. In some of their lumina, the cells have secreted a mucoid-like substance. The interstitial tissue consists of abundant fibrous tissue. At times, it resembles myxomatous tissue. These tumors are vascular, but are not usually as abundantly supplied with vessels as the other varieties. This type has been referred to in the literature as cylindroma. In the present series, the clinical course of this tumor has been considered the same as that of the other varieties of adenoma. We do not consider it malignant.

There are a few general histologic features about adenomas of the bronchus that deserve mention. Ordinarily, the surface of these tumors is covered by intact bronchial mucosa. In the specimens recovered at post-mortem examination, the tumors were rather sharply circumscribed, but there was no definite capsule. In a few of the adenomas of the present series, the cells exhibited a considerable number of mitotic figures. Extensive necrosis has not been observed, but occasionally there were a few hemorrhagic foci. Bone and cartilage have not been found as a part of the lesion in any of our cases.

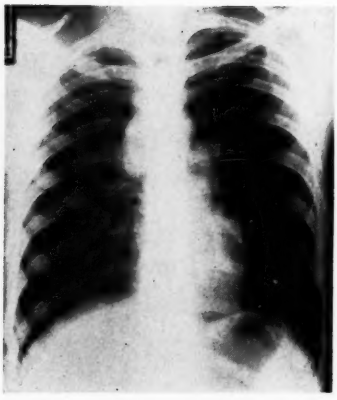


Fig. 3*A*.

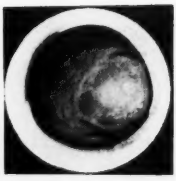


Fig. 3*B*.

Fig. 3*A*.—Roentgen study of chest of man, aged 21 years, who was examined bronchoscopically during 1942 and a large adenoma of the right bronchus found. There was a history of cough, occasional blood-tinged sputum, shortness of breath and wheezing respiration at irregular intervals and weight loss. A tentative diagnosis of tuberculosis was made in 1937. Roentgen studies of the chest made at that time revealed a tumor-like density above the right hilum which exhibited practically no change over the roentgen findings in 1942. (Film by Dr. Karl Kornblum.)

Fig. 3*B*.—Drawing exhibiting findings at bronchoscopy in the patient shown in Fig 3*A*. The right main bronchus was almost completely occluded by a rounded, soft, reddish tumor. Its point of attachment could not be determined. Following bronchoscopic removal of all available tumor from the right main bronchus, it was found that the upper lobar bronchial orifice was completely occluded by growth. Roentgen studies of the chest after removal revealed no change over that noted before bronchoscopy (see Fig. 3*A*). The tumor has its origin in the upper lobe bronchus and is not accessible to any form of endobronchial treatment. An external surgical approach has been recommended.

A critical study of the relation of the histologic structure and the clinical history was made in this series. There appears to be no relation between it and the clinical symptoms. The histologic picture, however, is important from more than an academic point of view. The failure to recognize the different types has been largely responsible for the variety of names applied to these lesions, from Mueller's first description in 1882 until the present. Of more importance is the tendency to confuse adenomas with carcinoma. Since

sometimes differentiation of the two lesions is extremely difficult, an appreciation of the variation in histologic structure is of great practical value in separating the two conditions.

While a pedunculated growth has been observed projecting from a main bronchus into the trachea, the point of attachment of the tumor usually is within the main or lobar bronchus and is unilateral. One case with unusual distribution of the tumor was observed.

CASE 1.—A woman, aged 45 years, was first observed during September, 1935. There was a history of cough for two years with occasional blood-tinged sputum. Recently she had developed dyspnea and pain in chest. Clinical and roentgen studies of the chest revealed an obstructive atelectasis of the left lung. At bronchoscopy performed during September, 1935, the left main bronchus was occluded by a rounded reddish mass. A diagnosis of adenoma was made by histologic study of tissue removed and in the course of several bronchoscopic treatments the endobronchial portion of growth was removed and the left lung reëxpanded. The point of attachment of the growth was on the posterior wall of the left main bronchus proximal to the bronchial orifice of the upper lobe.

The patient was advised to return in six months but failed to report until May, 1941. She was then extremely dyspneic and there was evidence of bilateral bronchial obstruction. At bronchoscopy the lower end of the trachea and both main bronchi were found partially obstructed by growth. This exhibited a granular appearance but was not ulcerated. Tissue removed at this time and at subsequent examinations revealed no change in the histologic appearance from the original tissue removed six years previously. She has been reporting for treatment every three or four months and now has a widespread distribution of tumor which involves the posterior wall of the lower end of the trachea and the orifices of the right and left main bronchi. No satisfactory explanation can be given for this peculiar distribution. The problem is to maintain the patency of both main bronchi.

The impression that adenoma does not become cancer is implied in the literature. In this series there is a single case in which cancer developed.

CASE 2.—The patient, a man aged 53 years, had been complaining of cough for four months. At bronchoscopy performed on Oct. 10, 1931, a rounded tumor was found occluding the left main bronchus. The histologic diagnosis was adenoma. He responded satisfactorily to treatment, and tissue from the bronchus frequently examined histologically revealed no change until November, 1936, when suggestive carcinomatous changes were noted. The patient died eighteen months later and the diagnosis of carcinoma of the bronchus was confirmed at autopsy.

On the basis of present knowledge, certain criteria must be considered in determining the plan of treatment to be instituted. These are the development of bronchial obstruction by the endobronchial portion of the tumor, the frequent occurrence of an extrabronchial portion of tumor which may produce bronchial stenosis, severe hemoptysis and the development of certain complications incident

Fig. 4.—Photomicrograph of the first histologic type of bronchial adenoma described in the text. (x100.)

Fig. 5.—Photomicrograph of the second histologic type of bronchial adenoma described in the text. (x100.)

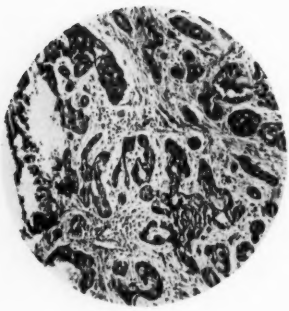


Fig. 6.—Photomicrograph of the third histologic type of bronchial adenoma described in the text. (x100.)

to interference with drainage; namely, suppuration, pulmonary fibrosis, bronchiectasis and pleural changes.

The slow growth of the tumor is conducive to the development of chronic bronchopulmonary changes. It is often difficult to ascertain the condition of the lung distal to the growth itself for the obstruction interferes with satisfactory lung mapping by the instillation of iodized oil. Ordinary roentgen studies of the chest commonly fail to give information concerning the extrabronchial portion of the tumor owing to atelectasis or chronic pulmonary changes incident to suppuration. In but three cases were we able to demonstrate by conventional roentgen examination the presence of an extrabronchial portion of the tumor (Fig. 1). The bronchoscopic appearances are often misleading for while one may remove the endobronchial portion and reëstablish the lumen, one can give no opinion concerning the presence or absence of an extrabronchial tumor.

The recent development of tomography has aided greatly in securing information concerning the relation of the tumor to the bronchus. In many instances in spite of extensive pulmonary changes it is possible now to demonstrate the presence of an extrabronchial mass indicating that the tumor is not limited alone to the bronchus. Obviously this additional diagnostic data will influence the plan of therapy to be instituted.

By bronchoscopic removal of the endobronchial portion one can usually reëstablish drainage of the lung distal to the growth. In several instances, however, it has been observed that there was compression stenosis of the bronchus apparently due to an extrabronchial mass of tumor, indicating that the endobronchial portion was not responsible alone for the bronchial obstruction. If it can be demonstrated conclusively on the basis of tomographic and bronchoscopic studies that the growth is endobronchial, bronchoscopic removal and electrocoagulation may be sufficient to reëstablish and maintain an adequate bronchial lumen. In this type of case therefore bronchoscopy will suffice (Fig. 7).

In the bronchoscopic removal it has been found very desirable, particularly in the pedunculated type of tumor, to employ the tip of the bronchoscope as a corer to remove the tumor rather than to resort to cutting forceps (Fig. 8). Bleeding commonly is encountered if removal is attempted and this interferes with the employment of cutting instruments. In our experience the coring method usually has not been followed by serious bleeding and has aided in

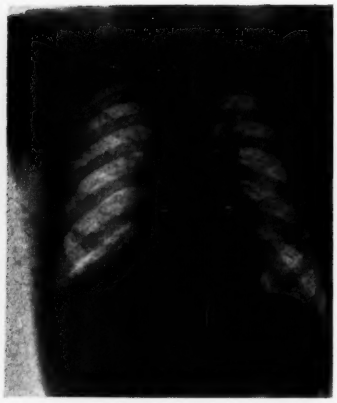


Fig. 7A.

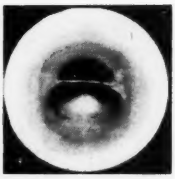


Fig. 7B.

Fig. 7A.—Roentgen study of chest of man, aged 41 years, revealing changes involving the lower lobe of the right lung which suggested retention of secretion with some collapse. The sharp definition of the lesion indicated that bronchial obstruction was present. (Film by Dr. W. F. Manges.)

Fig. 7B.—Drawing of bronchoscopic appearances of lesion observed in patient seen in Fig. 7A. A sessile purplish growth attached to the posterior wall of the right bronchus produced partial bronchial obstruction. Histologic diagnosis was adenoma. Growth was successfully removed by bronchoscopy and electrocoagulation. Patient, who has been observed annually for past eleven years, still is free from recurrence of growth and has no symptoms referable to the lungs.

more prompt removal of the tumor. In but two instances was it necessary to employ a bronchial pack.

It has been of interest in checking over the earlier cases treated by bronchoscopy to find that after years of freedom from symptoms and no recurrence of the growth endobronchially it has been observed that there was progressive narrowing of the bronchial lumen by either an intramural or an extrabronchial growth (Fig. 8).

Patients who have developed chronic pulmonary changes with bronchiectasis distal to the growth may secure remarkable relief from reëstablishment of ventilation and drainage of the lung. Whether one should proceed with surgical extirpation for relief of the bronchiectasis must be determined on the basis of the individual case. As a rule the bronchiectasis is not of the fetid type and commonly

Fig. 8.—Pedunculated portion of adenoma removed from left bronchus in case of girl, aged 19 years, employing the tip of the bronchoscope as a "corer". Bleeding was not marked and was readily controlled. Although extending into the left main bronchus producing obstructive atelectasis of the entire left lung, its point of attachment was the orifice of the left lower lobe bronchus. The endobronchial portion was successfully treated bronchoscopically; the extrabronchial portion ultimately produced obstruction of the lower lobe bronchus; bronchiectasis resulted and pneumonectomy was successfully performed.

reëstablishment of drainage aids remarkably in improving the patient's condition. Two illustrative cases are cited:

CASE 3.—A woman came under my observation during 1931 at the age of 21 years. There was found an adenoma producing atelectasis of the lower lobe of the right lung with extensive bronchiectasis beyond the stenosis. The tumor was treated bronchoscopically with diathermy and has not recurred. The patient is now married and has borne four children and is practically symptom-free (Fig. 9).

CASE 4.—A man, aged 45 years, was first observed in 1935. A clinical diagnosis of unresolved pneumonia had been made. An obstructing adenoma of the right lower lobe bronchus was found and successfully treated. Instillation of iodized oil revealed a bronchiectasis of the lower lobe of the right lung. He too is well without recurrence of growth.

There still is present bronchiectasis in both of these patients, but in the absence of bronchial obstruction there is absence of suppuration. Although lobectomy was recommended neither patient is inclined to submit to this plan of treatment.

In patients with bronchial obstruction, atelectasis, and chronic suppuration distal to the obstruction, it would appear that removal of a lobe or lung gives the best prognosis particularly if after attempts at bronchoscopic removal, the airway has not been maintained and the suppuration has not responded adequately. This may not be possible due to the patient's age and general physical condi-

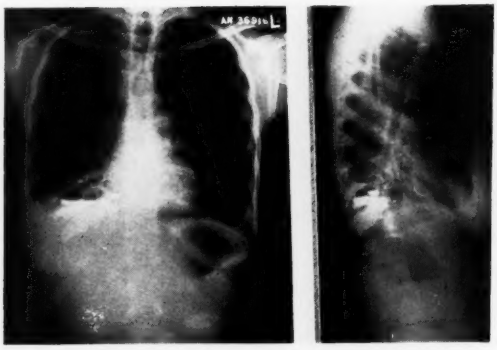


Fig. 9.—Roentgenograms of chest made following the instillation of iodized oil into the lower lobe of the right lung reveal extensive bronchiectasis. There has been no recurrence of the adenoma. Although lobectomy has been recommended for the bronchiectasis, the patient refuses treatment as cough and expectoration are negligible.

tion, for many of these patients have been ill with chronic pulmonary suppuration for many years before a diagnosis of adenoma is made (Fig. 10). It is only by correct diagnosis early before these changes have developed that a proper evaluation of the plan of treatment to be carried out can be made.

In our experience irradiation has been of no value. In the earlier cases erroneously diagnosed as adenocarcinoma intensive therapy was carried out without any appreciable change being noted in the growth itself.

In our series there have been four deaths which have resulted directly from the tumor. Two of these resulted from bronchiectasis, one from pneumonia which probably was dependent on bronchial obstruction and one from carcinoma which developed at the site of an adenoma of five years' duration. There were four deaths due to unrelated causes. One of the patients had been free from the tumor for eleven years. Seven patients are well and free from symptoms although there still is evidence of the tumor being present. Four patients have tumors with symptoms present; three of these have definite bronchiectasis and a fourth has paroxysms of coughing with

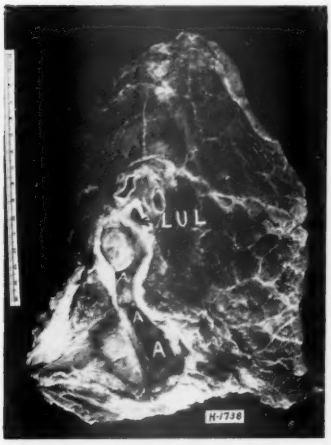


Fig. 10.—Specimen of left lung removed at autopsy in case of adenoma complicated by extensive bronchiectasis. A diagnosis of adenoma with complicating bronchiectasis had been made bronchoscopically and by the instillation of iodized oil. Surgical extirpation was refused and the patient went elsewhere to secure roentgen therapy and interstitial radiation. When seen eight years later he was practically in extremis and died from bronchopneumonia. The tumor is practically endobronchial, is contained in a greatly dilated bronchus, is attached to the posterior wall of the lower lobar bronchus being in contact with the upper lobar orifice (*LUL*). The lower lobe is atelectatic and fibrotic and consists almost entirely of a large cavity (*A*) which is continuous with the greatly dilated bronchus.

occasional infection distal to the tumor which is extrabronchial producing compression stenosis. This patient undoubtedly has bronchiectasis distal to the stenosis. Two patients have bronchiectasis without symptoms and an absence of tumor. They have been tumor-free for seven and ten years respectively; seven are tumor-free for periods varying from five to eleven years and also are free of symptoms. One has adenoma in both main bronchi and presents a hopeless problem. Three have recently come under observation. Two of these should be treated surgically for in one the tumor in the right upper lobe bronchus is inaccessible to bronchoscopic treatment (Fig. 3) and in the other there is a large extrabronchial tumor in the lower lobe of the left lung which is producing compression stenosis. The third patient is being treated bronchoscopically.

Surgical treatment has been recommended in six cases because of inaccessibility of the tumor to bronchoscopic treatment, the pres-

ence of extrabronchial adenoma or the occurrence of chronic pulmonary complications which cannot be relieved because of bronchial obstruction. Three have been treated surgically by lobectomy and two by pneumonectomy. There was one operative fatality. This patient had an empyema and was a poor surgical risk.

CONCLUSIONS

1. Adenoma of the bronchus is an epithelial tumor which probably is benign and is non-metastasizing. Its frequent occurrence as an extrabronchial and endobronchial tumor ultimately results in bronchial obstruction with chronic pulmonary suppuration, bronchiectasis and pulmonary fibrosis which if untreated tends to a fatal termination.

2. The early clinical and roentgenological findings are those of partial or complete bronchial obstruction. Later, changes in the lung distal to the tumor complicate the diagnostic problems. Ordinary roentgen studies usually fail to exhibit evidences of extrabronchial involvement. Tomography affords a valuable aid in determining the relation of the tumor to the bronchus.

3. Although earlier cases were diagnosed as adenocarcinoma, errors in diagnosis still are possible if based alone on histologic examination of inadequate material. The bronchoscopic findings are very important and should be considered diagnostically in conjunction with the biopsy studies.

4. The endobronchial method of removal may be adequate in the endobronchial type of growth. The common coexistence of extrabronchial tumor probably renders the method inadequate as a curative measure. Surgical extirpation is indicated in cases not accessible to bronchoscopic treatment, in cases associated with chronic pulmonary changes distal to the tumor and in cases of extrabronchial tumor.

1530 Locust St.

128 S. Tenth St.

LXXX

KODACHROME VISUALIZATION OF THE PHYSIOLOGY AND PATHOLOGY OF THE TRACHEO-BRONCHIAL TREE

PAUL H. HOLINGER, M.D.

CHICAGO

Endobronchial physiology is not satisfactorily understood. Mechanisms of secretion, the function of the bronchi as tubes, and the gross bronchial movements may be recorded, measured and analyzed to give concrete information to enlighten our knowledge of this subject. By means of a specially constructed endoscopic camera[1] the gross movements of the bronchi may be recorded in colored motion pictures to permit further study of these actions.

The film shows clearly the increase and decrease in the diameter of the bronchus which occur with respiration, as well as the lengthening and shortening of the bronchi with the respiratory cycle. By means of foreign bodies introduced into the bronchi of anesthetized dogs, various obstructive phenomena are illustrated. The mechanism of a wheeze, of check-valve and ball-valve obstruction, and of complete obstruction producing atelectasis are shown as they appear bronchoscopically.

To illustrate these various phenomena in the human being, the trachea and bronchi of patients with obstructing lesions are photographed bronchoscopically. Bronchiectasis, asthma, pulmonary adenoma, and bronchogenic carcinoma having distinct bronchoscopic features serve to demonstrate the physiologic and pathologic processes of the bronchi.

The camera devised for this endoscopic photography consists of a magazine loading 16 mm. camera and an *f*2.7 three-inch lens. Illumination of the small endoscopic field is obtained by directing the image of the ribbon of a ribbon filament projection lamp onto a 45 degree mirror which encroaches on the lumen of the endoscope. This type of axial illumination provides adequately the illumination essential to color photography. A specially constructed telescope

inserted into this system permits visualization of the field being photographed not only during the finding but also during the actual filming.

700 N. Michigan Ave.

REFERENCE

1. Brubaker, J. D., and Holinger, Paul H.: The Larynx, Bronchi, and Esophagus in Kodachrome, J. Biol. Photog. Assoc. 10:83-91 (Dec.), 1941.

LXXXI

FIBROLIPOMA OF THE LARYNGOPHARYNX

F. W. Davison, M.D.

Danville, Pa.

The patient, a 70-year-old man, applied for treatment on Aug. 18, 1941, stating that three years previously, while suffering from a temporary gastrointestinal upset, he had vomited a mass of tissue. He thought the tissue was a tonsil because an existing tumor mass of which he was aware was known to be adherent. He reswallowed it and the tumor continued to cause him no inconvenience. Two or three times during the past three years a temporary intestinal upset had caused him to regurgitate the mass and each time he noticed that it was a little larger. Aside from these temporary gastrointestinal disturbances he had had no symptoms and was ordinarily not conscious of the mass of tissue hanging in his esophagus.

Mirror examination showed a small pink rounded mass of tissue on the left lateral wall of the hypopharynx behind the larynx. When the tongue was firmly depressed, the patient gagged and regurgitated the long, smooth mass of tissue shown hanging from the mouth in Fig. 1. It was soft, was about one inch at its greatest diameter, and was apparently covered with squamous epithelium.

Using local anesthesia, the hypopharynx was exposed with a direct laryngoscope. The tumor mass was found to be attached by a pedicle one-half inch in diameter to the left lateral wall of the hypopharynx. The pedicle was transfixed with a needle-carried ligature and ligated. The pedicle was then severed by an electrode attached to the cutting current. A much smaller but similar tumor mass arising just mesial to the large one was then removed with the cutting current after its pedicle had been crushed. The large tumor mass after removal was eight inches in length and one inch in diameter at its thickest point.

The microscopic report by Dr. H. F. Hunt was as follows: "Section consists of a roughly circular piece of tissue which externally is covered with a layer of well-differentiated stratified squamous

Fig. 1.

epithelial cells. The stroma consists of fibrous tissue and some fat. Throughout the stroma are observed lymphocytes and plasma cells."

The patient had no postoperative bleeding or other discomfort and had a transuretheral prostatic resection four days later. Mirror examination of the hypopharynx six months later showed no abnormality other than a smooth white area of scar tissue at the site of attachment of the pedicle.

Geisinger Memorial Hospital.

Society Proceedings

SIXTY-FOURTH ANNUAL CONGRESS

OF THE

AMERICAN LARYNGOLOGICAL ASSOCIATION

The Sixty-fourth Annual Congress of the American Laryngological Association convened in the Marlborough-Blenheim Hotel, Atlantic City, N. J., at 10 A. M., May 25, 1942, Dr. Thomas A. Carmody presiding.

Ludwig's Angina

C. H. McCaskey, M.D.

Indianapolis, Ind.

Ludwig's Angina is a rapidly spreading cellulitis, beginning in the region of the submaxillary gland and extending to the floor of the mouth.

Dental infection, with or without trauma from extraction of diseased teeth, is one of the most frequent causes of Ludwig's Angina. Among other causative factors are injuries to the interior of the mouth, tonsillitis, peritonsillitis, Vincent's angina, facial erysipelas, scarlatina and diphtheria. The bacteriolgy is that of a mixed infection.

The symptoms are as follows:

1. A wood-like induration, sharply defined from the surrounding normal tissue.

2. The thrusting forward and upward of the tongue toward the palatal vault.

3. Severe dyspnea with the attending danger of laryngeal edema.

These symptoms are associated with the classical signs of infection of the phlegmonous type. The temperature is septic.

The fundamental requirement of treatment is radical surgery to decompress the phlegmonous tissues and relieve tension primarily and only secondarily to evacuate pus. The incision should be close to

and follow the edge of the mandible from the submaxillary gland to the midline. It is carried through the deep fascia. After this, further exploration is carried out by blunt dissection. If the sublingual tissues are tense and indurated, it may be necessary to divide the mylohyoid and, possibly, the geniohyoid muscles on the affected side.

Supportive treatment to combat the accompanying sepsis consists of fluids and glucose given intravenously, by proctoclysis or retention enemas.

The prognosis depends upon the promptness with which the condition is recognized and the thoroughness of the treatment.

DISCUSSION

DR. HENRY B. ORTON: I will bring out with slides the pull of the various muscles at the floor of the mouth and of the tongue.

The digastric pulls the mandible out and, if it is fixed at the mandible, it elevates the hyoid. The mylohyoid has a tendency to pull upward, the hyoglossus downward, the styloglossus upward, the stylohyoid upward, and the masseter muscle, below the level of the mylohyoid, downward.

The geniohyoid has a double action. Normally it elevates and pulls the hyoid bone forward but if it is fixed, then it depresses the mandible; so we have a pincer movement between the geniohyoid and the digastric. The geniohyoglossus has a triple action. The posterior portion protrudes the tongue, the anterior portion pulls it back and all of the musculature of the geniohyoglossus pulling together depresses it.

If we have a suprahyoid compression, which we have in Ludwig's Angina, this is what we find: The tongue is pushed up against the soft palate, the soft part of the uvula against the pharynx, the base of the tongue against the epiglottis and the epiglottis back to the pharynx. This is the danger zone in Ludwig's Angina.

The incision Dr. McCaskey spoke about is underneath the jaw and not only goes through the geniohyoid and the mylohyoid, but the digastric as well, thereby releasing the tension and possibly eliminating future trouble.

THOMAS E. CARMODY: There is one point to which I wish to call attention. Dr. McCaskey said that infection very seldom went through the gingival margin. I had a case a few years ago in which an osteomyelitis, following cleaning of teeth, broke the soft periosteum at the gingival margin producing an infection in the mandible. Later we had a true Ludwig's Angina.

I have seen several cases of that kind in which infection followed an injury. Dr. McCaskey spoke of them. Most of the injuries are due to fractures and then we have the infection on the inside of the mandible. The point that Dr. McCaskey spoke of is that the infection very seldom perforates the external surface of the mandible. Because the mandible is very much thinner on its inner surface, the infection drains easily to the lingual surface and the submaxillary region, where we get a true Ludwig's Angina.

DR. SAMUEL IGLAUER: The President has stressed the importance of history, and it so happens that I became interested in Ludwig and found an account of his life—that is, his surgical life.

It seems that when Napoleon invaded Russia, Ludwig had joined Napoleon's Army, and he got as far as that town which we read about so often, Smolensk, where he was captured by the Russians. He became ill there and probably had typhus fever. He finally was released or exchanged and got back to Stuttgart, where he recovered.

Later on, when Great Britain and Germany lined up against Napoleon, he joined the German Army and took part in the campaign against Napoleon, with whom he had been originally associated.

There are one or two points about Dr. McCaskey's paper I cannot quite understand. Why does he have to ligate the facial artery? I believe he mentioned that as a preliminary. According to my idea we are working under the jaw and do not particularly come into relation with the facial artery.

Another thing I would like to question is the bisecting of the geniohyoid muscle. I think Blair (Diseases of the Mouth and Jaws, C. V. Mosby Co., St. Louis, Mo., 1912, p. 338) points out that it is dangerous to cut one or both of these muscles because they hold the tongue forward; and if you should sever the geniohyoid completely, the hyoid bone would tend to fall back and aggravate the condition which Dr. Orton has described.

I would also like to know from Dr. McCaskey whether he thinks that this infection, as a rule, is chiefly under the mylohyoid muscle or above it.

It seems to me that we have two conditions, one in which the abscess is above the mylohyoid diaphragm, which might be called a sublingual abscess, and one which is below the mylohyoid, the true Ludwig's Angina.

I might say, furthermore, that Ludwig had good opportunities to study the pathology of this disease because, in his day, patients

were treated chiefly by poultices and most of them died; and so he had good autopsy studies.

DR. CHARLES D. BLASSINGAME: The important thing about Ludwig's Angina is true classification.

In the case that Ludwig described, the infection was above the mylohyoid and pushed the tongue up to the roof of the mouth. The abscess ruptured inside the mouth and the patient recovered without any operation. As I understand it, that is a definitely different condition from the infections below the mylohyoid muscle. In the latter cases one does not have an elevation of the tongue. The reason we still have a death rate of 50 per cent is because we do not make a distinction between those cases in which the infection is under and those in which it is above the mylohyoid.

If the tongue is raised there is an infection inside the floor of the mouth above the mylohyoid, and that is the type of case which Ludwig described. If the infection is below the mylohyoid, any surgeon can treat the case adequately. If it is above the mylohyoid and one does not cut through the floor of the mouth, the patient will die unless the abscess ruptures, as in Ludwig's case.

I think the factor of greatest importance in the Ludwig's Angina is that of pressure necrosis. The amount of pressure which can be generated by an infection in the floor of the mouth is quite tremendous.

I recall a patient who died by asphyxiation when the infection in the floor of the mouth generated enough pressure to push the tongue against the back wall. Much pressure, if confined, will cause necrosis and that is what happens in these cases, which Dr. McCaskey clearly brought out. The main factor in treatment is wide incision to relieve the pressure early, before necrosis takes place.

I recently had a case in which there was an infection in the floor of the mouth which I was draining. I thought I was getting adequate drainage, but the infection crawled over the border of the mylohyoid muscle and formed a lateral pharyngeal abscess. Then the swelling started to travel down the neck. This required external incision, after which the neck infection cleared up. The infection under the tongue, however, continued to drain pus profusely for about six days longer when a large mass of necrotic tissue came out from under the tongue. On the following day pus was no longer present and the patient promptly got well.

DR. FRANCIS L. WEILLE: I would like to call attention to Mosher's technic for an incision in this kind of case—an incision which drains both the neck and the floor of the mouth. His classical incision is medial to the submaxillary gland, which avoids touching the artery in the floor of the mouth. The area can thus be drained from the side.

DR. T. C. GALLOWAY: I am going to talk at some length on the question of anaerobes in soft tissue, as well as in bone. I think that in these cases of Ludwig's Angina more emphasis has been placed on the anatomical factors than on the question of the infecting organism.

In Ludwig's Angina the fascial limitations have been considered most important; yet is it not possible that anaerobes from the mouth determine to a great extent the nature of the process? The anatomic factor, of course, as Dr. Blassingame has shown, is important because in the region of the floor of the mouth the inflammatory swelling of the primary invasion shuts off the circulation and provides anaerobic conditions optimum for the spread of these particular organisms. Also the cure is brought about by wide incision which does not especially provide good drainage for a relatively unlocalized infection, but rather releases tension, restores circulation and allows access to the infected territory. The organism of Vincent is especially of importance.

At the County Hospital we have been giving arsenicals very early in these cases and we have reduced fully 50 per cent the morbidity.

DR. GEORGE B. WOOD: I thought we all believed that the Ludwig's Angina was a plain cellulitis of the floor of the mouth and entirely differentiated from any abscess concerning the submaxillary gland or the lymphatics of the neck.

It has been repeatedly said that the buccinator, the geniohyoid and the genioglossus muscles make a barrier against the spread of infection from the floor of the mouth into the neck. We can easily diagnose clinically whether we have phlegmonous cellulitis of the mouth or simply an inflammation of the submaxillary gland from, say, a calculus, by palpation of the floor of the mouth with our finger and by the elevation of the tongue.

My conception was that the term "Ludwig's Angina" was limited entirely to a phlegmonous cellulitis of the floor of the mouth which occurs in that fascial space between the geniohyoid and the

genioglossus muscles. Further, the asphyxiation does not come so much from the elevation of the tongue as it does from edema of the glottis. The infection spreads posteriorly and between the genioglossus and geniohyoid muscles directly back to the base of the epiglottis where it soon produces edema of the glottis itself.

In the cases which I have seen—they have not been many in number—when the asphyxia became marked the patients were distinctly hoarse, as you would expect from the edema of the glottis.

Dr. Mosher's operation of turning up the submaxillary salivary gland to reach the floor of the mouth is very scientifically developed without doubt, but it is easier for me to make a frontal incision. If one limits that incision from one facial artery to the other, it will give plenty of space and one does not have bleeding which amounts to anything. One can get directly into the floor of the mouth by cutting one of the geniohyoid muscles. One may also have to notch the anterior border of the mylohyoid muscle.

Dr. John F. Barnhill: Why do you need to cut the geniohyoid?

Dr. Wood: To get sufficient space to relieve tension, which is absolutely necessary. If there is much tension I think tracheotomy should precede the operation.

I was put in a great dilemma with one patient I operated on a number of years ago by having to do an emergency tracheotomy through a swollen, edematous neck, because the patient stopped breathing when I started to open up the abscess.

A thing I wanted to call attention to was this—maybe I am not right about it—that an abscess originating from the third molar tooth will not produce a Ludwig's Angina because it is back of the hyoglossus muscle. It is outside of the hyoglossus muscle. The hyoglossus muscle comes in front of the second molar tooth, and in order to get a phlegmon in the floor of the mouth the rupture of the tooth must be in front of the hyoglossus muscle.

Dr. Harris P. Mosher: When pus gets rampant in the neck it does not, in my opinion, pay much attention to those fascial demarcations.

The greatest satisfaction I have had in dealing with these cases —and most of mine have not been dental cases—has been with a liberal incision on the side of the neck, a wide incision to get under the submaxillary gland, which gives me control of the side of the tongue and I have a suspicion, which somebody who is not so ob-

sessed with his anatomy and his fascial limitations may sometime prove true or false, that some of these abscesses are frankly in the base of the tongue or are abscesses on the outside of the tonsil in the pharyngo-maxillary space.

I found some of them in which I went into an absolute pocket, either at the base of the tongue or outside of the tonsil, and I feel that there are more of those than have been put on record.

DR. C. H. MCCASKEY: I believe I said at the beginning that this type of Ludwig's Angina is a true, purulent lymphadenitis. I think there is just one thing to do: that is to get free and unlimited drainage.

In some of these cases we find no pus but we do have a tremendous pouring out of lymph after the incision, which seems to limit the tension.

If we wait until we find free pus then there is more danger of a septicemia. (Blassingame, C.D., Angina Ludovici, An Anatomical and Chemical Study, Trans. Amer. L. R. O. Soc., 1928, pp. 32-55).

My experience is that we get these cases late. I do not say very late, but when there is tremendous tension pushing up the tongue and spreading along the floor of the mouth. By doing a decompression of the neck we have some chance of saving these patients.

Dr. Mosher says he likes free and liberal drainage and that is what I like.

Infections With Anaerobic Streptococci With Special Reference to Cranial Osteomyelitis

THOMAS C. GALLOWAY, M.D.

EVANSTON, ILL.

Conventional descriptions of the spread of cranial osteomyelitis do not satisfactorily explain why this condition is perhaps more likely to arise from minor procedures and why the involvement of the compact bones of the face may be so dangerous. These observations, however, fit into the picture of anaerobic infection.

Nine cases involving cranial bones are reported in which anaerobic and microaerophilic streptococci were found. Four different pathological types of lesions occurred in this group. The excellent

response to treatment by zinc peroxide after the technic of Meleney in the later cases gives additional proof of the importance of these organisms.

Such organisms have probably been overlooked in many other infections in the field of otolaryngology.

DISCUSSION

DR. E. D. KING: Last fall I performed an antrum window operation on a young man, 35 years old, who had had chronic suppurating sinusitis and had been advised at various times to have a radical antrum operation done; but he finally compromised on a window operation. I operated upon him on September 20th and about ten days later he had a swelling of his face on the right side at the corner of his nose.

I observed that for about ten days. I discussed this case with Dr. Galloway, who described two or three of these cases and their treatment. My case seemed to be in the same class but I could not persuade any of those who were called into consultation to make any incision in that area because surgeons and otolaryngologists are loath to do that.

The swelling was quite typical, and as Dr. Galloway has described, it was indurated. It felt as though it were subperiosteal. Finally, after a month, when pressure made an opening, I could see the fistula and pus was seen exuding from it. The patient, after developing a tremendous cellulitis of the face, was persuaded to have the incision made; which was done with the cautery knife. A Shaw lateral rhinotomy was done and a large amount of pus was removed. There was no osteomyelitis, but bare bone could be felt with the probe.

From then on it was a tragic affair. This boy developed osteomyelitis of the nasal bones and the septum, osteomyelitis of the frontal bone, and finally subdural abscess and brain abscess. He died two months later.

During all this time, the surgeons were loath to make any incision; but finally, about six weeks after the original incision, when osteomyelitis could be demonstrated with the x-ray, an incision was made up through the center and the flaps were laid out and zinc peroxide was used; but by this time he had a subdural abscess.

I thought you might be interested to know the bacteriology. Dr. Altmeyer, who is a surgical bacteriologist at the Cincinnati Gen-

eral Hospital, sent me this note the other day: "We believe the responsible infecting agent was a micro-aerophilic hemolytic streptococcus which grew only anaerobically at first but after repeated subcultures became slightly aerobic."

That such an organism is known, Dr. Galloway has already told you. We had no difficulty in isolating this bacterium on many occasions during the course of the illness.

As Dr. Galloway advised, the sulfonamides were given in tremendous doses and one of the bacteriologists grew these organisms in the presence of sulfonamides in the media.

I think the thing which impressed me most about this case was the fact that the surgeons and those who were called into consultation were absolutely unwilling to make an incision. The other thing which impressed me very much was the fact that the sulfonamides were of no value at all in the handling of the case.

DR. V. V. WOOD: Have you any idea that there might be an exacerbation of these lesions when they were traumatized?

DR. MOSHER: This seems to be the age of miracles. The sulfonamide drugs came in and produced their miracles and now I am wondering whether the drugs used against anaerobes if dealt with properly, are not going to join the miracle class.

I cannot understand the surgeon being unwilling to make an incision in the presence of infection. Ever since Williams brought out this article, I have been interested in this subject and have followed it up a bit, but not to the extent Dr. Galloway has. So he has given me a lot of new information.

But, in one case of fulminating osteomyelitis which Dr. Shaw had recently, only a third culture produced an anaerobic streptococcus.

Dr. Galloway says you have to get the center of the sequestrum to get the organism. That makes the procedure a little difficult and the whole bacteriology of these infections of the anaerobic class of organisms seems to me very difficult. All our laboratories will have to learn a bit of new technic before we can be sure whether we have this organism or not.

I would like to ask Dr. Galloway, did I understand him to say that he went down to the periosteum, found pus under that in one of his cases and made no bone opening, but simply contented himself

with the local use of the zinc peroxide as far as he could uncover the bone?

To me it might make the procedure quite a bit complicated to follow the periosteum to its limit of denudation, so to speak, and to get the drug in.

DR. HOWARD C. BALLENGER: Since Meleney's work [F. L. Meleney, U. S. Navy M. Bull. 40:53 (Jan.), 1942], many other authors and investigators have emphasized three points about the zinc peroxide which Dr. Galloway mentioned. First, the preparation must be fresh to be active. Second, it is usually used in watery solution and must be in contact with all infected parts. Of course, this means that we have to do a complete surgical excision or exposure. Third, the dressing must be kept quite wet. I understand there is a preparation on the market now which is ready to use; that is, it is sterilized by dry heat.

I think some work is cited in *War Medicine* [Reed, G. B., and Orr, J. H.: Treatment of Experimental Gas Gangrene With Zinc Peroxide, War Med. 2:79 (Jan.), 1942] in which guinea pigs have been injected with ten times the lethal dose of these anaerobes and then zinc oxide has been used with good results, but sulfathiazole was used locally with about the same results. So it seems these two drugs could be combined and perhaps that would be of value. [Reed, G. B., and Orr, J. H.: Local Chemotherapy of Experimental Gas Gangrene, War Med. 2:59 (Jan.), 1942.]

I believe no results were obtained by inoculation, but the local use of the drugs did give results. Other sulfonamides apparently did not give any.

DR. THOMAS E. CARMODY: I would like to say that we have found around the jaws just what Galloway has called attention to; namely, the infection is under the periosteum as well as along the arteries. For instance, we find it under both the external and the internal periosteum, and there is no reason why we should not find it in other places.

We have also found that in many of these jaw cases we have the anaerobic culture problem.

DR. T. C. GALLOWAY: Dr. King's case was very interesting and, in discussing it, I rather predicted what the outcome would be if he was not allowed to operate.

McKenzie [McKenzie, D.: Jour. Laryngol. and Otol. 42:293 (May), 1927] said that in treating these cases of infection of the maxilla one should make a moat so that the infection could not get across into the frontal area, if one did that it would probably protect against any advance of the infection.

Dr. Wood asked about trauma. I believe the important thing to remember is that it is not the wide opening incision of a radical frontal operation which produces the trouble, but rather the minor operation which does not expose the organism to aerobic conditions.

In traumatizing, local death of tissue and destruction of some blood vessels has been caused, and I think that is the condition under which anaerobic infection can begin and advance.

As to the question of periosteal extension, I am inclined to think that the advance is likely to be under the periosteum at the very advancing border of the lesion.

In my case of a necrotic lesion I found that only by doing a lateral rhinotomy could I get the organisms from under the periosteum.

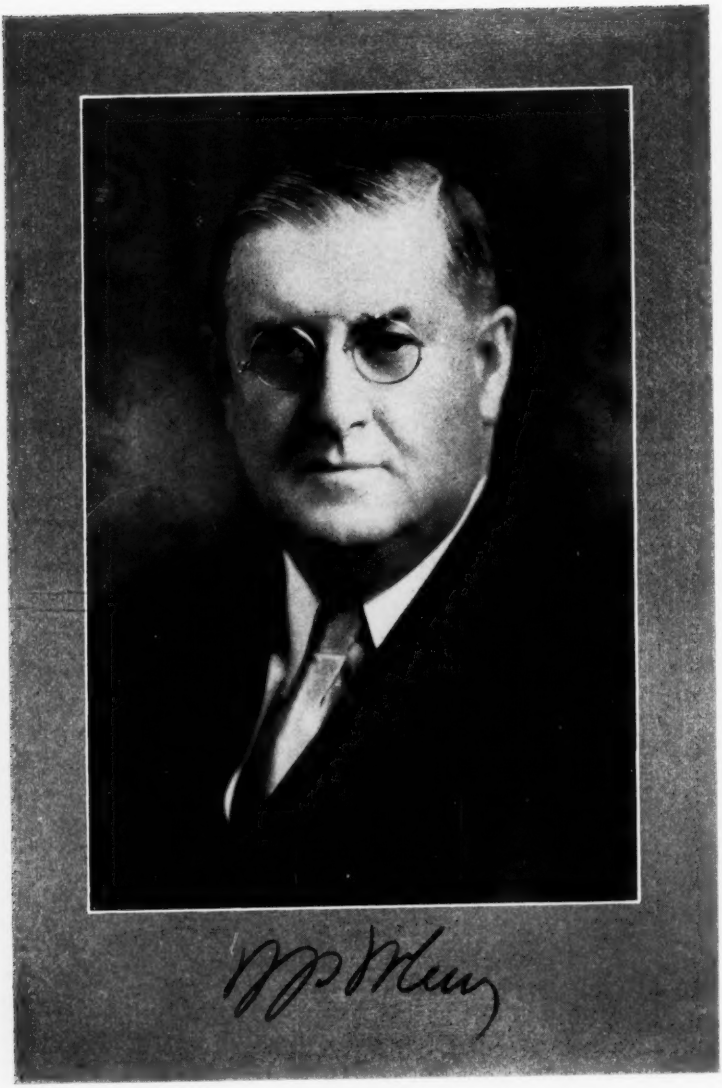
There is some difficulty in culturing these organisms. There are several ways of doing it: cultures may be gotten from the advancing border of the lesion or from the interior of the sequestrum.

It has been shown in mixed infections of the skin that if one draws pus into a pipet and allows it to stand for several hours, the staphylococcus will die and the anaerobic streptococcus will grow without being overgrown by the staphylococci. I think there are a good many technics for finding the organisms.

Dr. Mosher has always emphasized the importance of wide exposure. That is the first prerequisite, and if perfectly made, probably nothing further would have to be done.

There is one advantage in having a pocketed wound: one can put a tube in and irrigate with the zinc peroxide.

I infer that Dr. Ballenger speaks of war surgery. I think if one uses sulfathiazole under aerobic conditions, it is effective.



WILLIAM P. WHERRY

1880-1942

On June 13, 1942, there was taken from our midst, one of whom it can truthfully be said, "he was a beloved physician." William P. Wherry was born at Omaha, Nebraska, on June 16, 1880, of British stock. His preliminary education was obtained in Omaha, and he was graduated from the University of Nebraska Medical School in 1903. He spent most of his professional career in Omaha, and his practice was limited to Ophthalmology and Otolaryngology. He was for many years the Oculist and Aurist for the Union Pacific, and the Chicago and Northwestern railroads. He was a member of the Phi Rho Sigma and Alpha Omega Alpha medical fraternities. For many years, and at the time of his death, he was Professor and Head of the Department of Otolaryngology at the University of Nebraska Medical School.

One of the original diplomates of the American Board of Otolaryngology, he soon became a member of the Board, and from 1927 until his death was its very efficient secretary and treasurer. The gradual development of the American Academy of Ophthalmology and Otolaryngology as the greatest society of its kind took place during his tenure of various offices of this organization, Secretary of the Section on Instruction from 1920 to 1926, and Executive Secretary-Treasurer from 1926 to the date of his death. In addition to these organizations he was a member of the American College of Surgeons, the American Otological Society, the American Laryngological Association, the American Laryngological, Rhinological, and Otological Society, and numerous other local ophthalmological and otolaryngological societies.

He served as chairman of the Section of Otolaryngology of the American Medical Association in 1934 and at various times was president of the Omaha-Douglas County Medical Society, the Omaha and Council Bluffs Ophthalmological and Otolaryngological Society, the Nebraska State Medical Association, the Nebraska Academy of Oph-

DR. WHERRY Photographed During a Session of the American Board of Otolaryngology in Philadelphia a Week Before His Death.

thalmology and Otolaryngology, the Medical Society of the Missouri Valley, and the Sioux Valley Ophthalmological and Otolaryngological Society.

In addition to the multitudinous duties imposed upon him as a member and officer in these medical societies, he carried on a very busy practice, was an ardent stamp collector, and was keenly interested in athletics, especially those competitive contests involving his alma mater. Some medical men distinguish themselves by original research, and while his medical papers number twenty-nine and embrace the entire field of Ophthalmology and Otolaryngology, Bill Wherry distinguished himself by his exceptional administrative and executive ability. Truly in this capacity, a Commander-in-Chief has been taken from our midst and his shoes will not soon be filled.

He had a remarkable interest in the progress of medicine, particularly in his chosen field, a keen insight into the requirements of undergraduate and graduate training, and in the interlocking of the various fields of medicine. He had a warm sympathy with the problems of the younger men seeking an adequate training in the specialty. Many a tangle was brought to him for solution, and in spite of his large practice and arduous duties he never refused his help.

Dr. Wherry is survived by his widow, Eugenie Van Epp Wherry, two sons Walter and Robert, and a daughter Perdita L. Wherry Carr.

W. E. G.

PUBLISHED WORKS OF DR. WILLIAM P. WHERRY

Supraorbital Neuralgia—1906.

Vincent's Angina—1912.

Foreign Bodies at the Introitus of the Oesophagus, Reason for Care in Removal—1913.

Tonsillar Hemorrhage—1913.

Lymphatic Drainage of the Nose and Throat, Its Relation to Focal Infections—1916.

Hayfever and Its Relief Through Desensitizing With Pollen Extract—1915.

Focal Infections—1918.

The Relation of the Roentgenologist to the Otolaryngologist in Paranasal Sinus Study—1918.

A Rhinological Criticism of the Concept of the Average Internist Towards Sinus Study—1918.

A Study of the Lymphatic Disturbances of the Nose and Throat in Acute Aural Infections in Children—1919.

Oddities to Be Looked for in the Paranasal Sinuses—1923.

Acute Vacuum Frontal Sinusitis—1924.

Visual Disturbances From Paranasal Sinus Diseases—1925.

The Value of the Roentgen Ray in Rhinological Diagnosis With an Elaboration of the Oblique Method of Rhese—1926.

The Adaptation of Medicine in Industry.

The Use of Colored Lenses by Enginemen While in Service.

The American Board of Otolaryngology.

Chronic Otorrhea. Guest paper, Illinois State Medical Society.

The Teachers' Section, American Academy of Ophthalmology and Otolaryngology—1931.

Symposium on Graduate Education. Transactions of the American Academy of Ophthalmology and Otolaryngology, 1931.

The Immediate Postoperative Technic Incident to a Caldwell-Luc Operation—1932.

Eyes and Illumination—1932.

Paranasal Sinus Pathology and Focal Infections—1932.

Review of the Recent Research in Paranasal Sinusitis. Postgraduate Course, St. Louis, Mo., 1933.

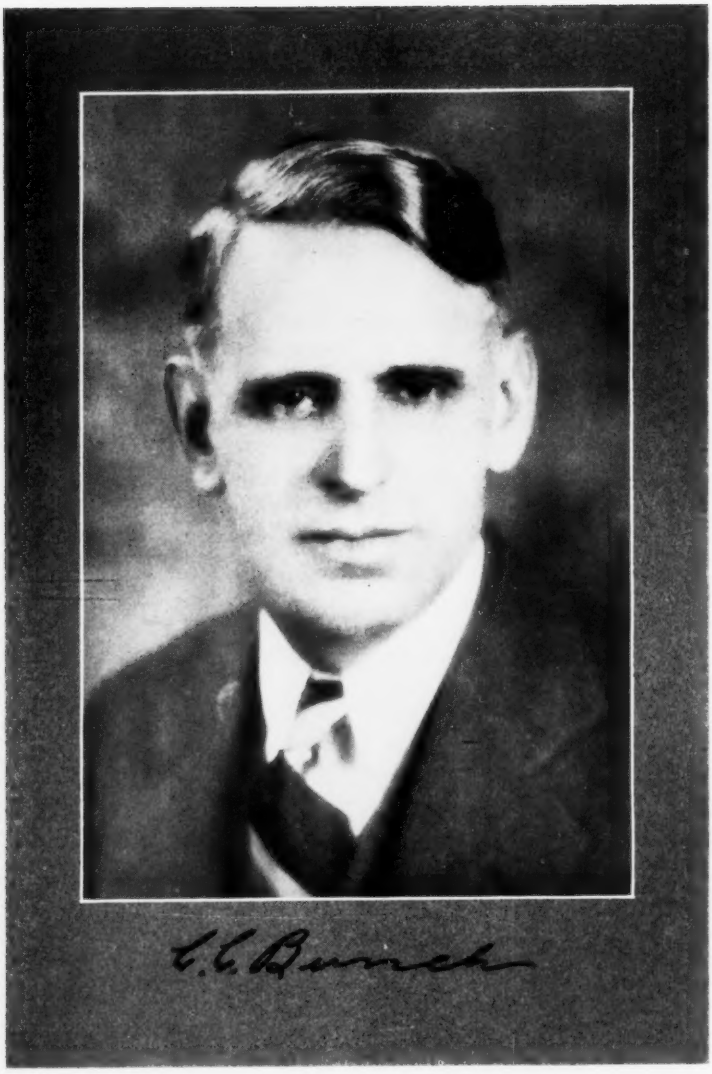
Registry of the Specialists—1933.

An Analysis of Many Thousand Miles of Engine Travel by a Railroad Ophthalmologist. International Journal of Surgery, 1933.

Graduate Education in Otolaryngology—A Viewpoint of the Examining Board for Otolaryngology. Chairman's Address, Section on Otology, American Medical Association, 1934.

Review of Candidates Appearing Before the Board for Otolaryngology—Report to the Advisory Board for the Surgical Specialties—1935.

My Son Studies Medicine. Address given before Philadelphia Laryngological Society, January, 1936.


E. E. Bunch

CORDIA CLARK BUNCH

1885-1942

Dr. Cordia Clark Bunch, Research Professor in Education of the Deaf at Northwestern University's school of speech, died June 14, while visiting his brother in Hart, Michigan.

Born in 1885 near Marshalltown, Iowa, Dr. Bunch was nationally known as a research otologist on the faculty of several universities and medical schools throughout the country. In 1919 he attracted attention as the inventor of the first audiometer—the Pitch Range Audiometer—which tested the hearing for a wide range of tones.

A student at Leander Clark College in 1902, Dr. Bunch received his B.A. degree from Iowa State Teachers' College, and his M.A. and Ph.D degrees from the State University of Iowa. In 1920 he was appointed research assistant in the psycho-physics of otology, and shortly afterwards was made Associate Professor of Otology at the University of Iowa Medical School.

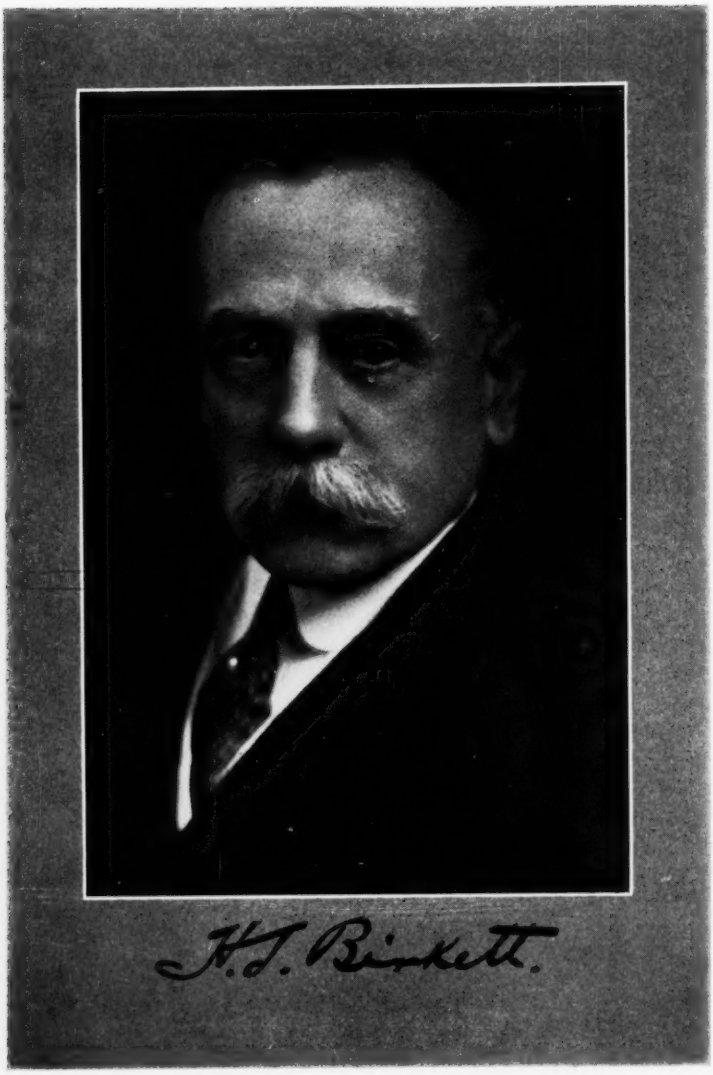
Dr. Bunch subsequently held teaching and research posts at Johns Hopkins, Washington University, and the Central Institute for the Deaf in St. Louis. He was also a lecturer in the American Academy of Ophthalmology and Otolaryngology and Associate Editor of the *Laryngoscope*.

His professional and fraternal memberships included the American Psychological Association; the American Otological Society; the Chicago Laryngological and Otological Society; the Chicago Speech Correction Society; the American Speech Correction Association; and the Gamma Alpha and Sigma Xi fraternities.

The author of more than a score of medical publications, Dr. Bunch contributed frequently to such journals as the ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY, the Archives of Otolaryngology, and the Journal of the American Medical Association.

He is survived by his mother, his wife Mrs. Lois Stoddard Bunch, and a brother Frank Bunch of Hart, Michigan.

I. F.



H. L. Birkett.

HERBERT STANLEY BIRKETT

1864-1942

Dr. Birkett was blessed with health, success, a long life, and a devoted family. He had the rare good fortune of celebrating his Golden Jubilee in Otolaryngology while still in active and vigorous practice. The occasion was suitably recognized by his confreres in 1939. Dr. Birkett was born in Hamilton, Ontario, the son of William Birkett, a merchant. He received his education from 1874 to 1881 at Forest House School, Chester, England, a fact of which he was always proud. He studied medicine at McGill University and graduated in 1886 with honors, before the age of 22, taking the Holmes Gold Medal for the highest marks throughout the whole four years. He was Senior House Surgeon for a year at the Montreal General Hospital, and after some months spent in general practice in Montreal, he went abroad to specialize in otolaryngology.

At McGill he studied under such men as Buller, Palmer, Howard, Osler, Ross and Shepherd. He always valued his experience in the Anatomy Room with Shepherd where he served as Demonstrator and then Lecturer for ten years. He was a member of the American Association of Anatomists. During his two and a half years special training in England and Europe, he worked under Professors Politzer, Chiari, van Schroetter and Schnitzler in Austria; under Professors Hartmann, Fraenkel and Kraus in Berlin; under Professor Fauvell in Paris; and in London he studied at the Golden Square Hospital under such men as Greville MacDonald, Bond, Mark Novell and Morell MacKenzie. He spent some time at the Royal Ophthalmic Hospital, London. On his return to Montreal, he became associated in practice with Dr. Buller, who according to the custom of the time, practiced Otology as well as Ophthalmology. At the outset, Dr. Birkett assisted Dr. Buller with refractions, etc., but after a time Dr. Buller practiced ophthalmology more exclusively and Dr. Birkett otolaryngology.

It is interesting to speculate as to what other factors contributed to his extraordinary success. He settled in Montreal away from family influence. As a student he was Organist at St. Mark's Church, Hamilton, and later at St. George's Church, Montreal. He rose to the top entirely through his own efforts. No detail was too small for his repeated consideration. This attention to detail permeated his whole life whether at work or play. In writing a paper, every detail

was carefully scrutinized, and every reference checked and verified at the source. The result is that Dr. Birkett's bibliographies are outstanding for completeness and accuracy.

He made more than eighty Atlantic crossings. The annual visit abroad was more than a holiday. Each time a new area was chosen and all the important medical centers therein were visited, but always with London as a base. He kept himself constantly up to date and was one of the best informed practitioners in the world.

Fearlessness and lack of false pride were outstanding qualities. When convinced he was right, he went ahead indifferent to public opinion. He had none of the small boy's false modesty which many of us carry into manhood, and which makes one hesitate to do the right thing because others may not do likewise or may criticize. In dealing with a patient he made up his mind what was the best course to follow, then he went ahead fearlessly, without further worry, and with no useless recriminations.

Meeting Dr. Birkett was always a pleasant occasion as his greeting was so warm and genuine. In the hospital the universality of his greeting cheered all alike. Outstanding victories of the football team organized by the Royal Victoria Hospital orderlies were the occasion of congratulatory cables from him while in Europe during the summer. In his care and devotion to patients he made no distinction between the poor and the rich, each received his best.

He was always dignified. He never scolded but he maintained strict discipline. He was intolerant of carelessness or inefficiency. Making hospital rounds with him was always a profitable experience. He had a keen intellect, a retentive memory, and was an ardent student. He was a perfectionist.

For many years he served as Secretary and later as Vice-President of the Canadian Medical Association and of the Montreal Medico-Chirurgical Society.

He was always a conservative surgeon. It was the conservatism of judgment, of knowledge and of experience, which stood him in good stead throughout his career. Dr. Birkett introduced bronchoscopy into Canada, also septal resection, the Killian radical operation on the frontal sinus, and radiography of the mastoids and of the nasal accessory sinuses. He was the first to perform in Canada thyrotomy for malignant diseases of the larynx in 1893.

He belonged to the leading international and national medical societies on both sides of the Atlantic and was a regular attendant at

meetings. He was an active member of the medical staffs and consultant to the following hospitals: the Alexandra Hospital for Infectious Diseases, the Children's Memorial Hospital, the Montreal Foundling and Baby Hospital. He was Otolaryngologist to the MacKay Institute for the Deaf, and latterly has been President and Honorary President of that Institute. He was a Life Governor of the Montreal General Hospital from 1887.

Similarly in organized medicine, he held many prominent positions. He was four times Vice-President of the Section of Laryngology and Otology of the British Medical Association. He was President of the Montreal Medico-Chirurgical Society in 1903, and President of the American Laryngological Association in 1907. He was made an honorary member of the Italian Laryngological, Rhinological and Otological Society.

He joined the Third Victoria Rifles of Canada as Surgeon-Lieutenant in 1887, and in 1894 he was a Surgeon-Major. He organized the first fully qualified regimental stretcher section in Canada. In 1909 he was President of the Association of Medical Officers of the Canadian Militia. It is fitting that at the time of his death he was Honorary Colonel of the Royal Canadian Army Medical Corps.

In 1914, he became Dean of Medicine at McGill University, a position which he held until 1922. In 1921, on the occasion of the Centenary of McGill University, he was given the degree of Honorary Doctor of Laws. In 1922, he was invited by the University of London to give the annual Semon Lecture in Otolaryngology. He was made an Honorary Member of the Scottish Otological and Laryngological Society in 1924, and of the German Ear, Nose and Throat Society in 1928. He was Honorary President of the Clinical Congress of Laryngology in 1919, and in the same year he was Vice-President of the Otological Section of the Royal Society of Medicine. He was a member of the American Bronchoscopic and Esophagological Society, and of the International Collegium of Otolaryngology. In 1913, he was Vice-President of the Otological Section of the International Congress of Medicine. He was President of the American Laryngological, Rhinological, and Otological Society in 1919, and President of the American Otological Society in 1922. In 1927, he was the first member to be a Guest of Honor of the American Academy of Ophthalmology and Otolaryngology. He was founder and first chairman of the Otolaryngological Section of the Montreal Medico-Chirurgical Society in 1933. In 1935 and 1938 he was Vice-President of the Pan-American Congress. In 1936 he was the repre-

sentative of the Dominion of Canada at the International Congress of Otolaryngology in Berlin.

His progress in Military Medicine was equally brilliant. In 1914, he organized and commanded No. 3 Canadian General Hospital (McGill), the first to be organized by any university and the first to take the field. He was promoted to full Colonel. In 1916, he was appointed Consultant Otolaryngologist to the British Armies in the Boulogne area. He was twice mentioned in dispatches by Field Marshall Sir Douglas Haig in 1916 and 1917. In 1918, he was made Assistant Director-General, Canadian Army Medical Services Overseas with the rank of Brigadier General. In 1917, he was created a Companion of the Most Honourable Order of the Bath (Military). He was also awarded the Colonial Officers' Auxiliary Forces Decoration in 1916, and the Jubilee Medal in 1935. He was Honorary Physician and Aide-de-Camp to the Governors-General of Canada, Lord Byng of Vimy and Viscount Willingdon, 1921-1931.

Dr. Birkett had not been in good health for the past eighteen months and he died on July 19, 1942, two days after his 78th birthday. There were occasions when he suffered intensely and yet through it all he never failed to be more concerned for those looking after him than for himself. Fortunately, his mind was clear to the last and he was able to enjoy the unselfish and tireless devotion of his wife and daughter. For Dr. Birkett death came as the fulfillment of an overabundant life, for his family and friends it is an irreparable loss.

W. J. McN.

PUBLISHED WORKS OF DR. HERBERT STANLEY BIRKETT

Seventh Lecture of the Somerville Course, before the Montreal Historical Society. Transactions of the Natural Society, 1889.

Diphtheria. Hare's "System of Therapeutics," 1890.

Hemiatrophy of the Tongue. Montreal Medical Journal, March, 1891.

Empyema of the Antrum of Highmore. Montreal Medical Journal, March, 1893.

Anomalous Cases of Primary Nasal Diphtheria. Montreal Medical Journal, June, 1893.

A Case of Sub-chordal Spindle-Celled Sarcoma and Its Successful Removal by Thyrotomy. New York Medical Journal, November, 1894.

Rhinoliths.

Salivary Calculi.

Perichondritis of the Larynx From an Unusual Cause. Transactions of the American Laryngological Association, 1896.

Diphtheria; Its Treatment and Nursing. Trained Nurse, April, 1897.

Address to the Graduates in Medicine at the Annual Convocation, McGill University. Montreal Medical Journal, May, 1897.

Oidium Albicans.

Carcinoma of the Larynx. By Birkett and Nicholls, Montreal Medical Journal, 28:343, 1899.

Foreign Body in the Naso-Pharynx. Montreal Medical Journal, June, 1899.

Dermoid Cyst of the Nose. Transactions of the American Laryngological Association, 1900.

Syphilis of the Larynx, Trachea and Bronchi. Buck's "Handbook of Medical Science," vol. 5, 1902.

Diseases of the Oro- and Naso-Pharynx, Posey and Wright's "Diseases of the Eye, Nose, Throat and Ear." Lea Bros. & Co., 1902.

Otomycosis Due to the Aspergillus Claucus. Montreal Medical Journal, May, 1904.

Lupus of the Oro-Pharynx and Naso-Pharynx. Transactions of the American Laryngological Association, 1904.

Further Report on Primary Lupus Vulgaris of Oro- and Naso-Pharynx. Transactions of the American Laryngological Association, 1905.

The Jubilee of Laryngology. Montreal Medical Journal, May, 1906.

Diseases of the Larynx. "Osler's System of Medicine," three editions, 1907, 1913 and 1928.

Early History of Medicine in the Province of Quebec. Presidential Address, American Laryngological Association; Transactions of the American Laryngological Association, 1908; and New York Medical Record, July, 1908.

The Value of Radiography in Mastoid Disease. Address given by invitation before the New York Academy of Medicine, 1908.

Demonstration of the Value of Radiography in Mastoid Disease given at the British Medical Association Meeting, London, 1910. The Lancet, Aug. 13, 1910.

Vasomotor Rhinitis. Address given by invitation before the British Medical Association; British Medical Journal, 1910.

Vaccine Treatment of Chronic Inflammatory Disease of the Accessory Sinuses of the Nose. Transactions of the American Laryngological Association, 1910.

Demonstration of the Value of Radiography in Mastoid Diseases given at the International Otological Congress, Boston, 1911.

The Education for the Specialist in Laryngology and Otology. Address given by invitation before the British Medical Association; British Medical Journal, August, 1912.

Geschichte der Ohrenheilkunde in Kanada. With Professor Adam Politzer in his book "Geschichte der Ohrenheilkunde, Ferdinand Encke, vol. 2, 1913.

The Organization and Work of No. 3 Canadian General Hospital (McGill) in France. Address given at the Annual Dinner of the American Laryngological Association; Transactions of the American Laryngological Association, 1918.

The Late Colonel John McCrae: Physician, Poet, Soldier. Address given before the American Laryngological, Rhinological and Otological Society; ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY, 29:98 (March), 1920.

The Transatlantic Development of Rhino-Laryngology. Semon Lecture, University of London, 1922.

Report of a Case of Carcinoma of the Larynx: Laryngo-Fissure. The Laryngoscope, August, 1923.

Buller, the Oculist; Politzer, the Otologist; and Lefferts, the Laryngologist. Transactions of the American Academy of Ophthalmology and Otolaryngology, 1927.

Chondroma of the Larynx. Canadian Medical Journal, 17:703, 1927.

Diseases of the Tonsil. In Jackson and Coates' "The Nose, Throat and Ear and Their Diseases", W. B. Saunders Co., 1929.

The Late Dr. Grant Stewart and Sir William Osler's "Way of Life." Address given to the second year medical students, McGill University; Medical Undergraduates' Journal, 1:2, 1932.

Lipoma of the Larynx—Intrinsic in Origin, Festscrift für Professor Doctor Ino Eubo of Tokio, 1934, Journal of Laryngology and Otology, 49:11 (Nov), 1934.

Contact Ulcer. Discussion on a paper presented by Chevalier Jackson at the Annual Meeting of the American Medical Association, Atlantic City, N. J., 1933.

The Early History of British Otology—1910.

A Short Account of the History of Medicine in Lower Canada. Address delivered before the 39th Annual Meeting of the Nu Sigma Nu Fraternity, Toronto, 1938.